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THE POLYGRAPH AS AN AID IN DIAGNOSIS OF CARDIAC CONDITIONS IN CHILDREN*

GODFREY R. PISEK, M.D., AND T. HOMER COFFEN, M.D.
NEW YORK

That there has been no startling advance in our methods of physical diagnosis since the days of Skoda and his pupils will probably not be denied. These men left us a rich heritage, and we are only now, through the use of scientific instruments, blazing new paths and adding valuable record findings to those of the unaided senses.

The use of the polygraph with adults has proved of great value in correlating physiologic and clinical data. It has removed that element which we speak of as the "personal equation," and instead gives records which are true for all time, and only require correct interpretation. These records have stimulated closer study of the heart and the circulation, so that the anatomist, physiologist, pathologist and clinician have all profited thereby and developed newer conceptions of the intricate cardiac mechanism.

The studies of Gaskell, who showed that the heart muscle itself possesses characteristic tone, that it can originate its own stimulus and conduct it to various parts of the heart, explained many of the phenomena recorded in polygraphic tracings. For example, the physiologist now knows that the auricle contracts an appreciable, measurable interval before the ventricle, and that the stimulus which produces cardiac contraction arises at the mouths of the great veins, spreading down successively over the right auricle, thence through the bundle of His and its branches to the ventricles.

Further, it has been shown that a dissociation between auricle and ventricle can be produced experimentally by destroying the tissue at the auriculo-ventricular junction, which contains special muscle and nerve fibers, and which seems to be a "connecting pathway" for the contraction stimulus.

With the polygraph we can show definitely whether the cardiac rhythm is normal; if normal, whether the arrhythmia is due to respir-

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atory or other influences; whether ventricular contraction follows auricular contraction as it should; whether the excitability of the heart is normal. With this instrument it is possible to study the functions of the cardiac muscle, namely, tonicity, rhythmicity, contractibility and conductivity. Thus far, the function of conductivity is best understood, and we find it disturbed in the usual forms of pulse irregularity. A study of the polygraphic tracings tells whether the irregularity is due to heart block, fibrillation of the auricle, or extra-systole, and, if the latter, which one of the three varieties of extra-systole is present. All these facts are of the greatest importance, for we have at hand by this means, in certain cases, just as precise knowledge (of the cardiac function) as one obtains by finding the malarial parasite in the blood-cells.

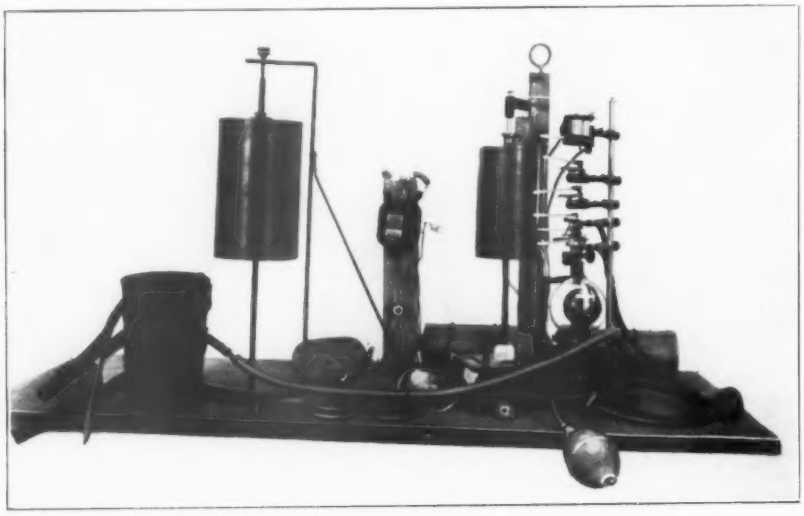


Fig. 1.—Modified Sphygmograph. With this instrument long tracings may be taken of brachial artery, apex, respiration, jugular vein and time (in 1/10 second).

With these facts in mind, we undertook the application of these methods in cardiac conditions in children, to determine (1) if satisfactory tracings could be made from children; (2) if information additional to that obtained in the physical examination was possible; (3) if this information could be utilized in prognosis and treatment.

The instrument used in our work was our modification of the Erlanger sphygmograph with a Hirschfelder attachment (Fig. 1). This apparatus allows a simultaneous tracing to be made (1) of the pulse — known as the sphygmogram; (2) of the heart — cardiogram; (3) of the venous pulse — phlebogram. These three tracings, when studied in

relation to each other, become much more valuable than when taken alone, even with the finest instrument.

The sphygmogram gives information relating to the movements of the left heart, the rate and rhythm of the heart, and the variations in pressure within the artery.

The cardiogram gives the tracings made over the precordium, or apex, and gives exact data concerning the heart movements, and aids in defining the cardiac systole. It should not be forgotten that the cardiogram does not record the pressure within the ventricle, but does give information as to the displacement of the heart in relation to the chest wall. It further indicates certain physiologic events which are

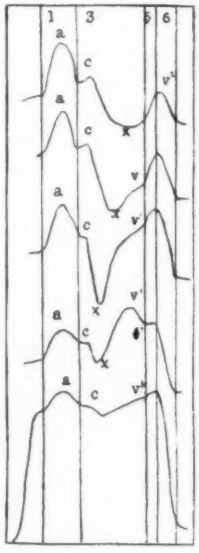


Fig. 2.—The Normal Phlebogram (Hay). Diagram from actual tracings showing the variations in the records of the auricular type of venous pulse. The upper phlebogram is normal, the lowest shows the marked effect of free tricuspid regurgitation in increasing the engorgement of the right auricle. Note the gradual increase of the wave *v* at the expense of the auricular depression *x*. The portion *v'* of the ventricular wave *v* occurs in the phlebogram before the perpendicular line 5.

helpful in determining pathologic states of the heart. In other words, the cardiogram gives information regarding the duration of the systole and diastole, the beginning of ventricular systole, and indicates the period of the action of the aortic valves and the opening of both cuspid valves. Sometimes the normal type of cardiogram becomes inverted — where there is such marked hypertrophy and dilatation of the right ventricle as to force the left ventricle out of position on the chest wall. How valuable such information may be to the clinician need not be

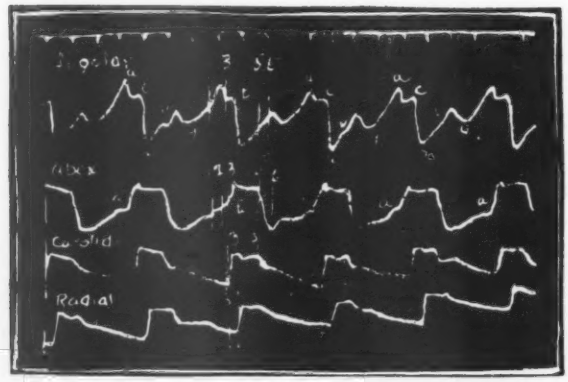


Fig. 3.—Normal tracings from jugular vein, apex, carotid and radial arteries recorded simultaneously (Mackenzie). The line above the jugular tracing is the time in 1/5 second. Note the waves, *a*, *c* and *v*. The beginning of the wave *c* occurs just 1/10 second before the rise (due to ventricular systole) in the radial as shown in line 3 in radial and jugular tracings. The *a* wave, due to auricular systole, begins about 1/5 second before *c* as shown by line 1, in the jugular tracing. This is during the diastole of the ventricle as shown by the falling line in the radial tracing at this time. The beginning of the wave *v*, occurs usually at the beginning of the rise in the radial. Its fall, however, marks the opening of the auriculo-ventricular valves and is coincident with the dicrotic notch of the radial tracing. The jugular pulse varies considerably, but the waves *a*, *c* and *v* can be measured off as has been explained.

Fig. 4.—Digitalis heart-block, in a boy 15 years old. After stopping digitalis the heart-rate became normal. At A′ in the jugular tracing is evidence of auricular contraction, while there is no contraction of the ventricle. A, right jugular, B, brachial, C, apex curves.

emphasized here, since he may be able to note on subsequent records in convalescence the gradual return of the left ventricle to its place, as shown by a positive cardiogram.

The phlebogram is of these three records the most important, as by representing the variations in venous pressure (when taken in the neck),

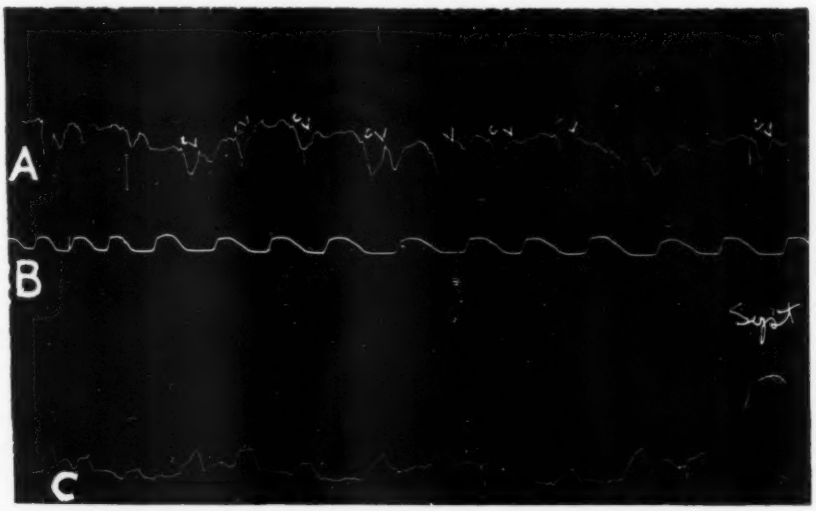


Fig. 5.—M. M., aged 10 years; respiratory arrhythmia; mitral insufficiency. Large jugular tracing, third heart sound. "H" wave in jugular. A, right jugular, B, brachial, C, apex curves.

Fig. 6.—E. M., aged 15 years; normal heart; respiratory arrhythmia. Note the large jugular waves in upper tracing.

it indicates the activities of the right side of the heart. While in some instances the interpretation of these tracings has led to controversial theories, in the main observers agree, and at all events the records stand ready for any advance in this field of medicine.

In reply to the first problem which we undertook to answer: Can tracings be obtained satisfactorily from children? In our series of cases,

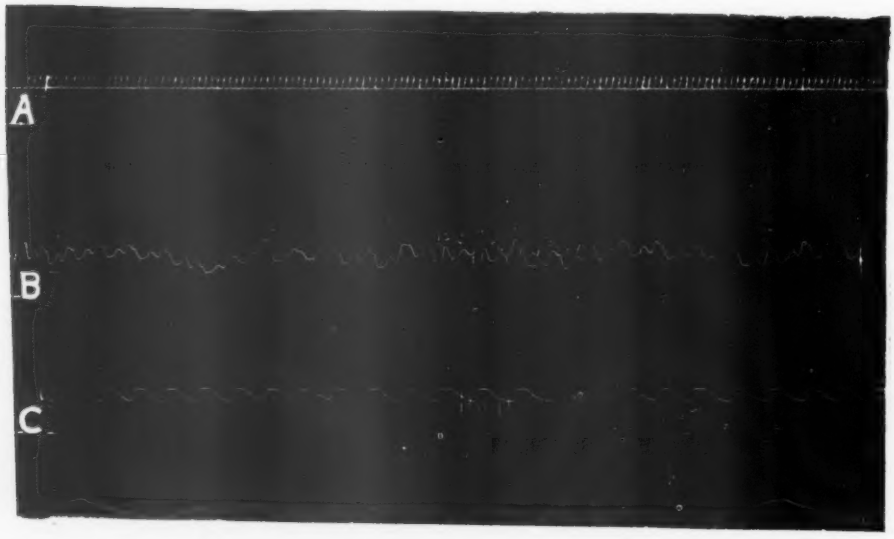


Fig. 7.—B. W., aged 4½ years. Congenital pulmonary stenosis. Note the large jugular waves. A, time, 0.1 second; B, right jugular, C, brachial curves.

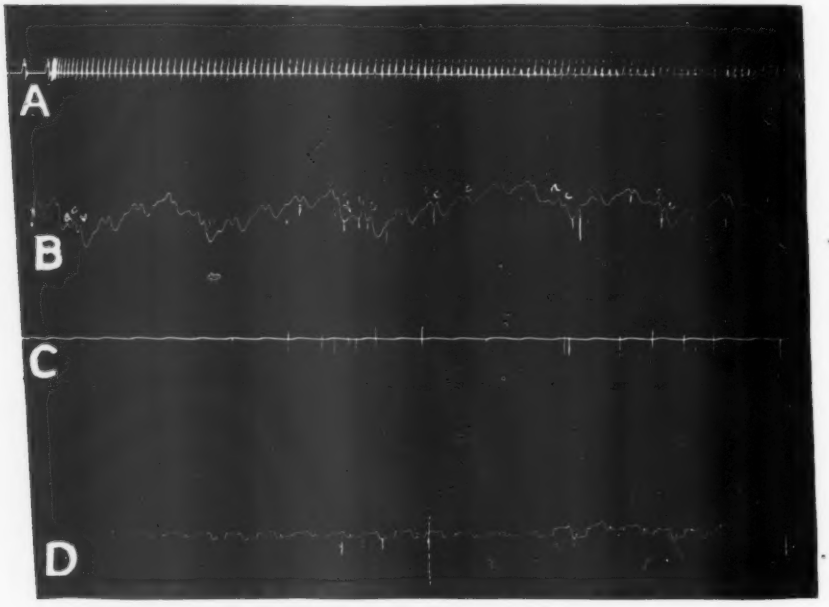


Fig. 8.—M. K., aged 5. Mitral insufficiency—uncompensated. Note large jugular waves and protodiastolic wave in cardiogram (associated with "third heart sound"). A, time 0.1 second; B, right jugular, C, brachial, D, apex curves.

the ages of the children varied from 4½ to 15 years, and all were suffering from cardiac lesions, either congenital or acquired. In each case we obtained satisfactory tracings from the brachial artery, precordium and the jugular bulb. With only slight modification of the appliances to suit the smaller anatomical areas we were able to make records without difficulty. For examples of well-marked jugular tracings in children see Figures 5 to 14 inclusive.

Before taking up the second question, or the value of tracings taken in children, it may be well to review briefly the anatomic and physiologic essentials on which interpretations are made.

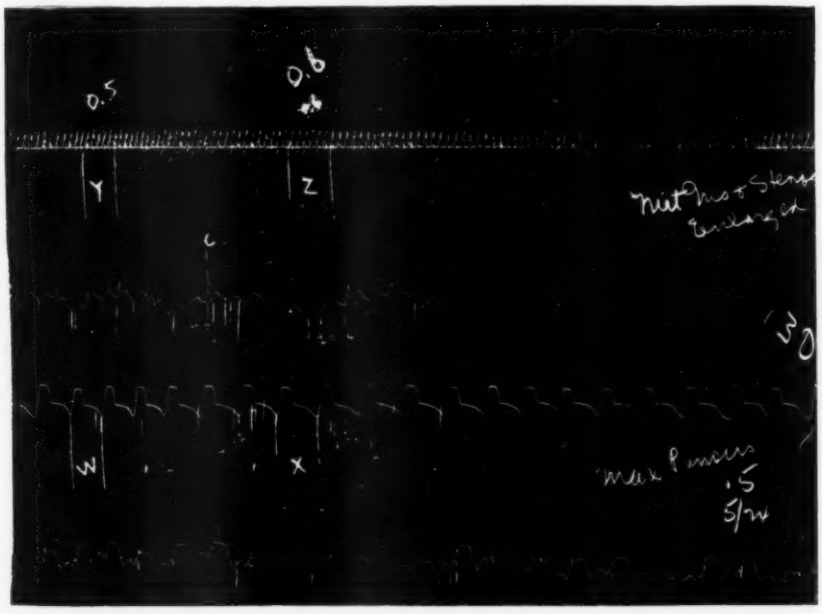


Fig. 9.—M. P., aged 5. Mitral insufficiency and stenosis; cardiac enlargement. Note respiration effect on cardiac rhythm; increased frequency with inspiration, decreased with expiration. Cardiac cycle W, 0.5 sec., cycle X—0.6 sec. Note protodiastolic wave "p" in cardiogram (associated with third heart sound) and corresponding wave "h" in jugular.

The right jugular bulb, which is found to be about one inch outside of the sterno-clavicular articulation, is the point over which the cup is placed to obtain the venous tracing. The right internal jugular, innominate vein, and superior vena cava, extend almost in a straight line from the heart. The pulsations are best obtained by lowering the child's head and placing it in such a position that there is a relaxation of the sterno-cleido-mastoid muscle. The best point to obtain the tracing from the jugular vein is about three-quarters to one inch external to the right sterno-clavicular articulation.

The normal venous tracing shows three distinct waves, called the (a), (c) and (v) waves (Figs. 2 and 3). The (a) wave occurs early in diastole of the ventricle, as can be seen by placing the hand over the apex and watching the time of this rise in the jugular pulse. The wave, however, is the result of the auricular contraction.

Following the (a) wave, a negative phase is recorded, termed (x), caused in greater part by the diastole of the ventricles, but this is interrupted by the (c) wave which is synchronous with the carotid pulsation.

The "a-c interval" is almost constant in point of duration, i. e., one-fifth of a second in the normal heart — and indicates the time of passage

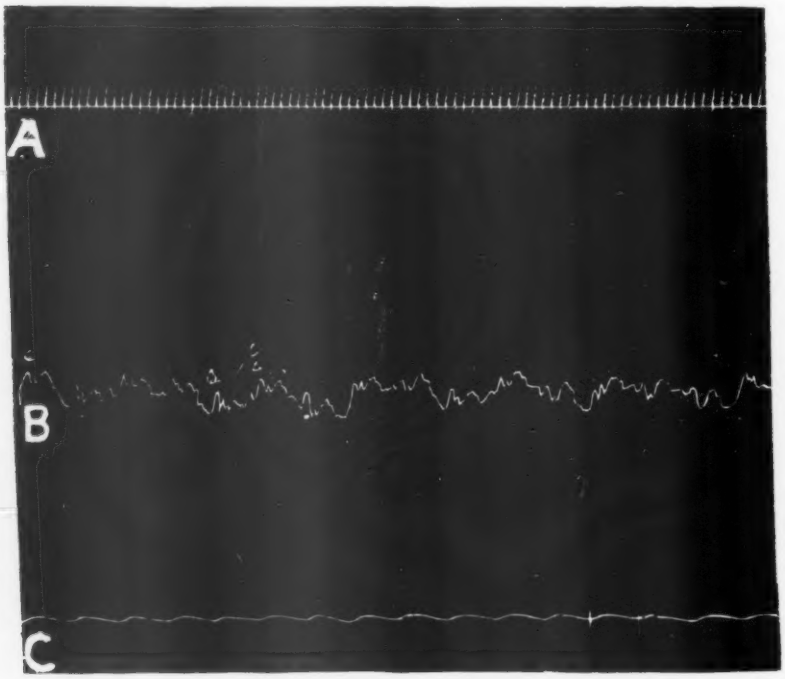


Fig. 10.—T. S., aged 7 years. Mitral insufficiency—uncompensated. Note the large jugular waves (B). A. time 0.1 second. B. right jugular. C. brachial curves.

of the contraction stimulus through the "conducting pathway" (Bundle of His).

The third wave (v) occurs synchronously with the systole of the ventricle. It is increased in size by any factors which tend to more rapidly fill the right auricle, and hence the veins.

A second negative diastolic phase (y) follows the (v) wave and records the emptying of the cervical veins. The importance of noting this wave is seen in cases of engorged right heart with over-distended veins in the neck. In these instances it is found practically absent.

To analyze a tracing from the jugular vein, one must compare it with a tracing taken at the same time from the brachial or radial artery (see Fig. 2). With a pair of compasses, place the left point on the first vertical or base line in the radial tracing; place the other point at the beginning of any rise; then move back the right leg of the compass one-tenth of a second, as indicated on the timer above; then place the left leg on the base line of the jugular, and the right point will fall on the beginning of the rise of the (c) wave. This should be the time for (c) in the jugular tracing, for it occurs one-tenth of a second earlier in the jugular than in the radial. Then measure one-fifth of a second to the left of this (a) wave, which should fall on "a." The wave following (c) is (v).

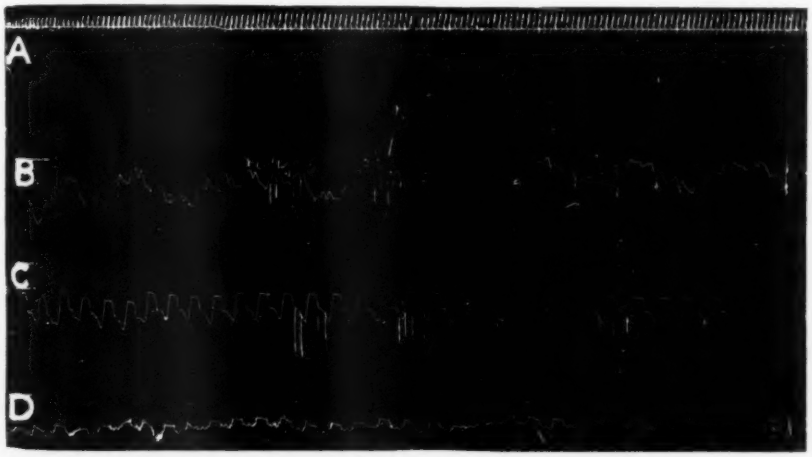


Fig. 11.—L. B., aged 12 years. Neurotic heart. Note character of jugular and cardiac tracings. A well-defined "p" is present in the cardiogram, with an occasional "h" wave in the jugular tracing. A, time 0.1 second; B, right jugular; C, brachial; D, apex.

In interpreting any polygraphic tracing, it is advisable to consider the functions of cardiac muscle, particularly that of conductivity. Cases of Stokes-Adams disease have recently been reported in adolescents, and undoubtedly cases of partial heart-block occur with severe cardiac lesions.

Normally, the a-c interval is of short duration. If the function of conductivity is interfered with, then the passage of the stimulus through the auriculo-ventricular bundle (or bundle of His) is delayed, causing the a-c interval to be lengthened. This form of cardiac arhythmia is found in acute ulcerative endocarditis, and not infrequently in cases of heart-block which have come to necropsy actual destruction of the bundle of His has been found.

If the lesion in this auriculo-ventricular bundle is so extensive as to cause complete "blocking" of the stimulus, then ventricular contraction does not follow that of the auricle, as it normally should, resulting in what is known as "heart-block." The dissociation between auricle and ventricle may be absolutely complete (the auricle beating independently of the ventricle and vice versa) or the ventricle may beat to every second, third or fourth auricular contraction.

Digitalis is often indiscriminately prescribed to children because they "have a valvular lesion." The polygraph teaches that it should never be prescribed for any individual whose a-c interval is prolonged, since it then only depresses the function of conductivity and accentuates any arrhythmia which is present. Such an instance from our series is presented in Figure 4. The patient was a boy of 15 admitted to the hospital with marked decompression. He had been taking digitalis in large doses — 15 minims of the tincture four times a day. On the seventh day the pulse, which had been regular, showed marked arrhythmia. The tracing, Figure 4, was taken on the eighth day, and shows typical heart block. The digitalis was then stopped, and seven days later his pulse was regular. The cause of such irregularities can best be recognized by the polygraph. Here we have a condition which, if not recognized, may become serious. Ordinary methods of physical examination do not reveal the real condition.

Of the other functions of cardiac muscle, that of stimulus production was found particularly interesting in our series of tracings. In *every case* we found a quickening of the pulse with inspiration and a slowing with expiration. This is known as respiratory arrhythmia (Figs. 9, 5 and 6). That it is caused by irregularly produced stimuli is shown by the fact that ventricular contraction follows auricular contraction normally; in other words, that there is no disturbance of conductivity, an important fact to recognize. The cardiac muscle in neurotic children is particularly predisposed to this disturbance.

No evidences of the function of excitability were noted in our records. This was contrary to our expectations, since the child is apparently susceptible to abnormal or irregular stimuli in other portions of its organism. We concluded, therefore, that the myocardium in the young does not exhibit any extra systoles, even in rapidly beating hearts.

Another point of interest in our study was the occurrence of the "third heart sound." This is accompanied by characteristic waves in the cardiogram and phlebogram (Figs. 5, 8, 9, 10). The significance of this is as follows:

In some cases the existence of this extra heart sound was not noted on the preliminary physical examination, and it was only recognized after its appearance on the tracing had specifically called our attention to its presence. Thayer states that it can be heard at the apex in about

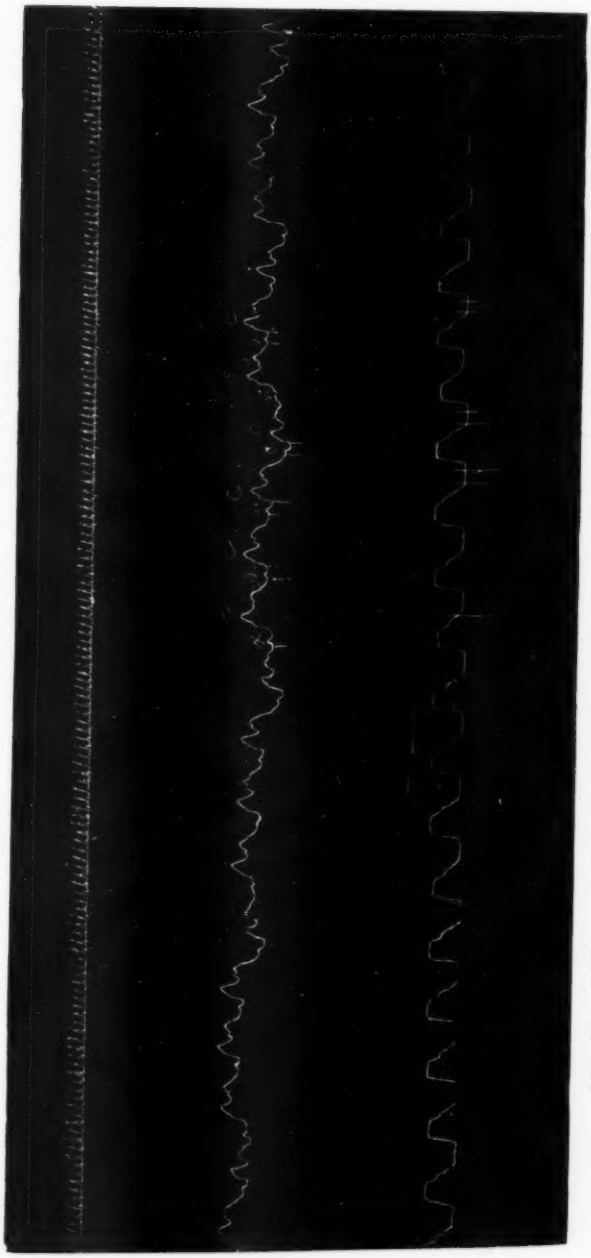


Fig. 12.—G. S., aged 12 years. Mitral stenosis. Note large jugular waves (upper tracing).

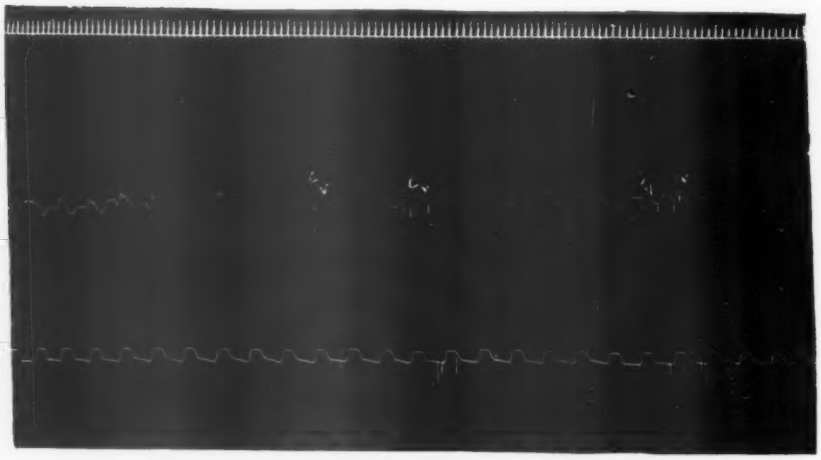


Fig. 13.—E. B., aged 13 years. Mitral insufficiency; overacting heart with slight hypertrophy. Note the excellent jugular tracing with normal *a c v* waves. Auricular type.

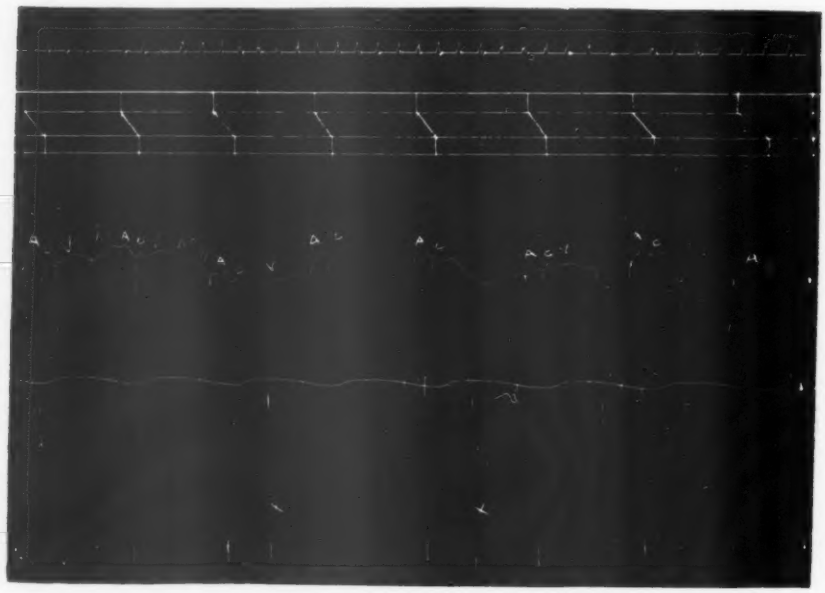


Fig. 14.—T. S., aged 6½ years. Mitral insufficiency and aortic insufficiency. Diagram represents passage of stimuli through auriculo-ventricular bundle. Normal

30 per cent. of normal individuals, when they are lying on the left side. In the first decade it occurs in 58.9 per cent. of cases.

This protodiastolic wave in the cardiogram is associated with an extra wave (h) appearing soon after the (v) wave in the jugular tracing (Figs. 5, 8, 9, 10) and therefore occurs in the early filling of the heart; in other words, at the very beginning of diastole. At this time the mitral and tricuspid valves are closed by the elastic recoil of the heart walls. Hirschfelder believes that it is due to the sudden snapping together of these valves. This wave may be pronounced whenever there is a ventricular distention — as in dilatation following hypertrophy, in stenosis of the mitral valve, and in aortic insufficiency, in which there is found to be an increase of intraventricular pressure. It is always accompanied by a wave designated as "p" on the cardiogram.

CONCLUSION

When making a physical examination of a patient with heart disease, perhaps the least important for the patient is the determination of the precise valve involved. The examiner should endeavor to determine the exact power of the heart to respond, to study the functionating ability of the myocardium and obtain data of the cardiac activity. These facts, in conjunction with the physical findings as to size, possible dilatation, or organic lesion, enable the physician to form a much more precise opinion as to the prognosis, while his treatment, at least, is based on scientific facts and can be controlled by further observations.

The results of our study may be summarized as follows:

1. Tracings satisfactory for diagnosis can be obtained in children.

2. The polygraph offers a means of accurate diagnosis which is not afforded by ordinary methods of examination, and should be used in addition to these — it will not replace them.

3. The information obtained from polygraphic tracings is of value for prognosis and treatment. Digitalis heart-block should be suspected where the pulse becomes irregular under the use of this drug.

4. Respiratory arrhythmia was found in our series with surprising frequency. This is said to be physiological, but we are led to suspect that it may frequently be misleading, and that the physician may suspect serious cardiac weakness. This was illustrated in cases of scarlet fever, diphtheria and measles studied by us at the Contagious Hospital. The "irregular hearts" were shown to have marked respiratory arrhythmia. We have yet to find extra systoles or auricular fibrillation in children.

The use of the polygraph will not stultify physical findings; on the contrary, more careful examinations will be made, for its use enhances their value and interprets observations which have thus far been unexplained.

36 East Sixty-Second Street—121 East Thirty-Eighth Street.

THE PHYSICAL EVIDENCE OF THE THYMUS[1]

CARL BASCH, M.D., AND ADOLF ROHN, M.D.
PRAGUE

Translated by A. C. Soper, Jr., M.D., Chicago

Recent experimental work in the physiology of the thymus has cleared up matters in two directions, namely, the relation of the organ to growth and to the condition of the bones, on the one hand, and to the electrical excitability of the nervous system, on the other.

When one completely extirpates the thymus of a young dog in the first weeks of life, there occurs as a consequence not only a checking of growth, but a condition in which the bones become softer and more flexible and show a smaller callus after an artificially produced fracture, than is seen in a control animal of the same brood. If one tests the electric stimulation of a young dog over the median nerve or the cerebrum, before and after the extirpation of the thymus, one can demonstrate after extirpation a gradually increasing hyperexcitability, which may be raised to the same degree as that produced by extirpation of the "epithelkorperschen." By injection of soluble calcium salts this hyperexcitability can be again reduced to normal.

Originally these experiments were based on an article by Basch[2] read at the Natural Philosophy Convention in Karlsbad in 1902, entitled "Extirpation of the Thymus Gland;" later communications appeared in the *Jahrbuch für Kinderheilkunde*, 1906 and 1908. Naturally these articles met with criticism from various sources, but were completely substantiated.

If we wish to refer to articles demonstrating the relation of the thymus gland to the osseous system, we note the work of Cozzolino[3] and Ugo Soli,[4] that of Lucien and Parisot,[5] of Sommer and Floercken (Würzburg),[6] of Ranzi and Tandler (Wien),[7] and the recent work of Klose and

1. Originally published in Deutsch. med. Wchnschr., 1911, xxxvii, 1843.

2. Basch, K.: Wein. klin. Wchnschr., 1903, No. 31; Jahrb f. Kinderh., 1905; and 1908; Monatschr. f. Kinderh., vii; Ztschr. f. exper. Path. u. Therap., 1905; Lotos, lvi.

3. Cozzolino: La Pædiatria, 1903; Congresso di Med. Int. Padova, 1903.

4. Soli, Ugo: Arch. ital. de biol., 1907-9; Mem. R. Acad. Modena, 1909; Policlinico, 1907.

5. Lucien and Parisot: Arch. d. méd. exper., 1910, p. 98.

6. Sommer and Floercken: Berl. klin. Wchnschr., 1908; Phys. med. Gesellsch., Wurzburg, 1908.

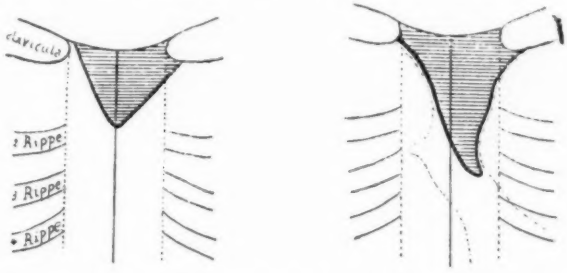
7. Ranzi and Tandler: Wien klin. Wchnschr., 1909, No. 27.

Vogt (Frankfurt)[8] entitled "Clinical and Biological Features of the Thymus."

Klose and Vogt in their comprehensive research into the extirpation of the thymus in young dogs not only showed the bone changes, but were the first to prove the nerve irritability, and were able to prove undoubted organic changes as well.

Gebele[9] and Hart and Nordmann[10] in their researches also obtained undoubted symptoms of loss of function. Among the later workers in the newer physiology of the thymus only Rudolf Fischl[11] failed to find these after thymus extirpation, and came to the conclusion that the thymus has no rôle in extra-uterine life. The negative result of this author is, however, as Basch has shown, due to the fact that the experiments of Fischl were arranged in a faulty manner, and the elementary rules of thymus research neglected.

Both the changes mentioned — the change in the development of the osseous system and the increase of nerve excitability in an animal deprived



Figs. 1 and 2.—Diagrammatic representations of the thymus gland, after Blumenreich.

of its thymus — depend simply and clearly on a uniform cause — alteration of metabolism, a point recently demonstrated by Basch[2] and by Bracci.[12]

The biology of the thymus is intimately connected with the function of the thyroid as well as the epithelial cells, with which it is histogenetically connected, and belongs to the same great group of the "branchiogenous organs."

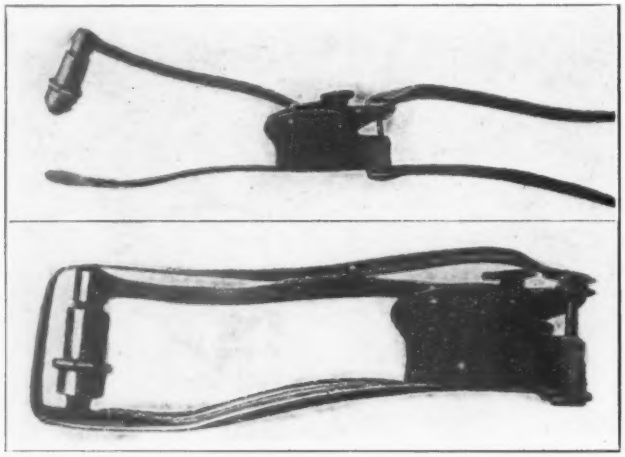
The relation of the thymus to osseous development and to nerve irritability is by no means a specific peculiarity confined to this organ, however. What gives the thymus its peculiar difference from the other organs which have an internal secretion is the fact that its function is

8. Klose and Vogt: Beitr. z. klin. Chir., lxix.
9. Gebele: Arch. f. klin. Chir., 1910.
10. Hart und Nordmann: Berl. klin. Wchnschr., 1910, No. 18.
11. Fischl, Rudolf: Ztschr. f. exper. Path. and Therap., 1905; Monatschr. f. Kinderh., vi.
12. Bracci: Rev. Clin. Paediat., 1905.

only transient, and that besides its correlation to the glands with internal secretion, it is to an extraordinary degree related to the lymphoid apparatus of the body; conclusions as to this are possible only after an exhaustive research. When a thorough research into the physiology of the thymus has been taken up, it is to be hoped that especial attention will be given to the differences existing in the living.

To find such a method, or to prepare the way for it, was the first object of our work, and for this purpose the use of the material in the Franz Josefs Kinderspital was offered. Robert Blumenreich[13] was engaged in a similar work in 1900, and the results of his work, which we shall next review, were reported in *Virchow's Archives*.

Blumenreich used as material for his work mostly the cadavers of the Berlin Pathological-Anatomical Institute, and made exact drawings from



Figs. 3 and 4.—Specially devised percussion instruments for outlining the thymus.

over fifty-five cases. Following the directions of Sahli, he lightly percussed with the finger the thymic area, drew on the skin the areas of relative and absolute dulness, and marked them internally with blackened needles driven into the chest. Then by dissection he proved the relation of the percussion outline to the actual position of the thymus. Also, in fifteen of his fifty-five cases he had the opportunity to percuss the thymic area both before and after death. Of the fifty-five cases, twenty-two were infants between 1 month and 1 year old; the rest were children up to 9 years.

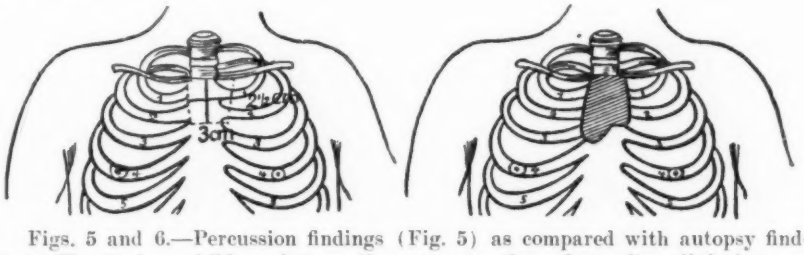
Blumenreich found that up to the end of the fifth year there was an unmistakable area of dulness over the thymus which in general presented an irregular triangle, whose base was the sterno-clavicular articulation.

13. Blumenreich: Virchow's Arch. f. path. Anat., clx.

and whose rounded apex lay at the level of the second rib, or occasionally just below it, while the sides reached over the edge of the sternal line. The larger part of the area of dulness lay generally on the left side, and the form of it varied but little within the age limits mentioned. After the sixth year of life the frequency with which dulness was found increased (Figs. 1 and 2).

If one compares the area of thymic dulness on a living child as drawn by Blumenreich, with the position of it in the cadaver, one sees that by this method the glands bordering on the lungs, as well as the "lingual-formed" lobes of this organ in the living, do not appear to view; and for more complete investigation a research which will also include these parts is necessary.

If one deliberates as to which is the most suitable method to completely solve this question, one naturally chooses the Roentgen ray in the first place, since it possesses an advantage over other methods in being most free from the danger of errors. Since Blumenreich's publication the Roentgen ray photography of the thymus has been used very extensively by Holzknecht[14] in 1901, Hochsinger[15] in 1903, and by L. Rehn[16] in 1906.



Figs. 5 and 6.—Percussion findings (Fig. 5) as compared with autopsy findings (Fig. 6) in a child aged 4 months; necropsy three days after clinical examination.

Rehn, in roentgenizing the anterior thorax, noted a downward movement of the gland in inspiration and an upward movement on expiration. According to Hochsinger the thymus shadow normally impinges on the heart shadow like the narrow neck of a bottle, and forms a band covering the shadow of the vertebral column. According to Holzknecht a completely developed thymus shows, by sagittal rays, a triangular shadow in the midthorax like that of an enormously dilated heart or a pericardial effusion. He ascribes it to the thymus, whose shadow obscures that of the large vessels and the heart.

In frontal and oblique rays, the usually clear retrosternal space is covered by the thymus shadow. No Roentgen pictures are given in the article.

14. Holzknecht: Fortschr. a. d. Geb. d. Röntgenstrahlen, 1901.
15. Hochsinger: Wien. klin. Wchnschr., 1903.
16. Rehn, L.: Arch. f. klin. Med., 1906.

Desiring to form for ourselves an independent judgment as to whether it is possible to obtain a clear shadow of the thymus in a Roentgen photograph, we prepared photographs of a young dog having a relatively large thymus, before and after extirpation, by means of instantaneous and time exposures, and a lead screen. In order to mark the exact location of the extirpation we sewed up the sternal cut with silver thread; nevertheless we were unable to find in the photographs any marks which could be definitely declared to be thymus shadows.

Next we took instantaneous pictures of an anatomic preparation of the sternum of a young dog, one exposure with thymus attached and another of the sternum alone; we could neither find any sign of a thymus shadow, nor see that the clear picture of the sternum was at all obscured by the underlying thymus.

Also, the thoracic photograph of two young children of the ages of 2 and 6 months gave negative results in respect to thymus shadows. The taking of further pictures induced us to regard the question of the clearness of the thymus shadow as still unsettled, and to speak very reservedly of our ability to give an instantaneous and unmistakable conclusion of the size of the thymus. Perhaps the instantaneous photography of Dessauer has given the progress desired; in an article by Alweus[17] a striking result in this line is seen in the accompanying thorax photographs of a child with pertussis.

But even if the experience with the Roentgen ray had been more satisfactory than was the case, better results would be found in refining and improving the older physical methods of percussion and auscultation of the thymus, since much smaller and simpler apparatus is needed for demonstration, and therefore it can be much more easily used in daily practice.

Since the position of the thymus is much more hidden than that of other large glands, such as the liver or spleen, one must admit, *a priori*, that the differentiation of this organ from its surroundings is much more difficult. Even if it is feasible now to get a more exact idea of the width and length of the thymus by percussion than before, this outlining of its size is to be accepted with caution, since by percussion alone no idea is to be had of an important factor — the thickness of the thymus. But in spite of this fact, we should not cease in our endeavors to perfect this method of estimating the size, since only in this fashion can the clinical foundation of pathologic study of the organ be undertaken.

If one looks for a contrasting problem, take the one of outlining the heart. It, as well as the thymus, is an organ characterized by its being free from air and yet in part overlapped by the air-filled lungs. Yet the heart affords a far easier outline for demarcation by percussion, having

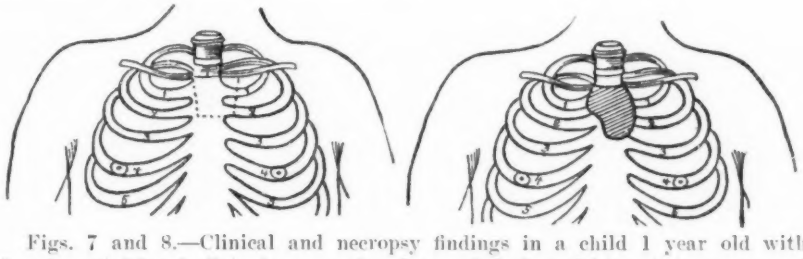
17. Alweus: München. Wchnschr., 1911, p. 737.

a firmer structure, being filled with fluid, and covered only by the thin ribs and intercostal muscles.

The thymus is covered by the thick sternum and rests on the air-filled trachea, by which any jarring may change the percussion sound, and the thymus edges are almost continuously covered by lung tissue. In emphysema of the lungs, in which the heart is generally more obscured by the dilation of the lungs, and held in a rigid thorax, the percussion more nearly approaches that of the normal thymus.

A short time ago the use of auscultatory percussion for outlining the heart in emphysema was suggested by Bock[18] in Munich, who recommended the use of a specially constructed stethoscope, a procedure which compares very well with the Roentgen-ray outlining.

The method urged by Bock consists in this, that his stethoscope as originally designed permitted a gradual muffling of the sound and was put on the middle of the sternum; the sound was shut off about one-half and the area was percussed by an assistant.



Figs. 7 and 8.—Clinical and necropsy findings in a child 1 year old with chronic nutritional disturbance and subnormal body-weight. Autopsy several months after clinical examination.

According to Bock the person examining with the instrument in the manner directed — shutting off the half — is able to hear those vibrations which are usually lost in the deeper parts of the thorax, while those arising in the superficial parts, such as the edges of the lungs, aid the return of the deep percussion note.

The conventional percussion of the upper thorax with one's finger as recommended by Sahli and Blumenreich,[13] afforded us a too meager result, as the finger seemed too thick an instrument to use on the thorax of an infant, so we invented a little "stockel-plessimeter" for light percussion of the thoracic area, and confined ourselves to auscultatory percussion. In order to make it possible for one person to do this auscultation and percussion, and to make the technic as exact and uniform as possible, Basch constructed an instrument which duplicates the mechanism of the percussing finger but is lighter and can be easily used with one hand. It touches only about a quarter of a centimeter of surface, and can easily be moved across these small areas laterally as well as longitudinally. The

18. Bock, H.: München. med. Wchnschr., 1908, No. 11.

half shut-off stethoscope is held over the mid-sternum with the other hand, and the percussion note heard by the one who makes it.

The use of this new instrument shows that it is possible to obtain a more definite and exact outline of the anterior thoracic organs than by earlier methods such as the finger. In contrast to the triangular area of dulness over the thymus pictured by Blumenreich, we found, by direct and by auscultatory percussion, an area somewhat larger than that which has been before demonstrated. It is probable that the instrument will be found to be of value in general percussion, as with it the cardiac area of a young child with yielding thoracic walls may be easily outlined.

By careful percussion of the anterior thorax with the instrument shown in Figures 3 and 4 the area of absolute thymus dulness is obtained and drawn with a colored pencil on the skin. This drawing we regard as the first sketch of the area of dulness, to be perfected by further examination. In many cases the relative dulness is obtained by auscultatory percussion, especially toward the left side of the thorax. In general the results from the auscultatory percussion agree with those of the precise instrument.

Besides this, we have used a method to outline the lateral areas of the thymus, described by Bianchi and A. Smith[19] a short time ago — the so-called friction method. For we found the original method devised for heart outlining, by which the phonendoscope is set in the area of absolute dulness and the adjacent areas stroked with a brush or the finger, very complicated and not well chosen. The friction method we used in this way:

An open, sound-magnifying phonendoscope was placed in the thymic area, a small bristle brush drawn diagonally over the sternal area, and attention paid to the point where the scraping sound was weakened or deadened. Only in cases in which the thymus extended beyond the sternum could we find a change in the scraping sound — where it seemed to be of considerable thickness; thus a further means of estimating the thymus was found.

A simple physical experiment illustrates the technic of the friction method. If one takes a cardboard box, on the bottom of which a heap of sand is put, and holds it in an oblique position, when the outer side is scratched with a brush one can locate the edge of the sand by the cessation of the scratching sound if this has a depth of only a centimeter. Marking the border found and opening the box at the same angle, one is convinced of the correctness of the marking.

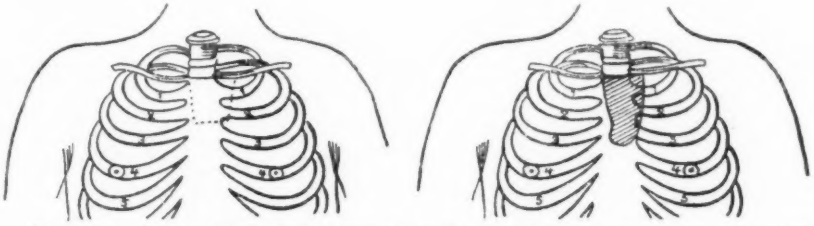
A similar phenomenon is observed in the friction examination of the thymus area. If one lays the open bell of the phonendoscope over the thymus area, first to the left and then to the right, and with a brush strokes the skin in an oblique direction, one hears the transmission of the

19. Smith, A.: (Refers. to Bianchi) Kongr. f. Inn. Med., 1900.

scratching sound as long as the brush is over the air-filled lungs. The sound is deadened or silenced on the spots where lung tissue ends and the thick thymus underlies the sternum. In children under 2 years, in whom the thymus is flat or small, the method is useless, as well as on the cadaver, in which instance the lungs have collapsed.

By this method we examined in the out-patient clinic of the hospital a number of children aged 6 to 18 months, and later up to 3 and 4 years. Beyond the fifth year no sure thymus dulness could be demonstrated. For the greater part of our study we used, as far as possible, well-nourished and developed children with only minor diseases, in order to have a picture of the physiologic condition of the thymus. But we also collected a considerable amount of material for the pathologic study, regarding which we will speak later. A majority of the findings it was possible to prove by autopsy; these were naturally the ones most valuable for instructive deductions.

As a rule each of us worked independently of the other, and drew his findings on a diagram of the anterior thorax. After some preparatory



Figs. 9 and 10.—Clinical findings (Fig. 9) confirmed at necropsy (Fig. 10) in a child 1½ years old, well nourished, dying of descending croup.

trials necessary to make one familiar with the peculiarities of the method, the results obtained were uniform. Usually we took only a few cases each day, because the experiment was conducted with strained attention and therefore produced fatigue. The methodical procedure was the following:

The child, undressed, was first laid on a sofa in a flat position, and a roll put under its neck and shoulders to throw the upper thorax more forward, lifting the chin upward. Then with our percussion instrument, whose surface spanned perhaps a quarter of a centimeter, the anterior thorax was methodically percussed, with the lightest and most uniform strokes, and point by point in exact rows the area to be examined was studied in a lattice work of small squares. The dulness found by direct percussion was marked on the skin with a blue pencil, and next the half-deadened stethoscope of Bock placed in the middle of the sternum and fixed with the left hand, and the thymic area again percussed with our instrument. Alterations shown by auscultatory percussion were drawn on the figure.

Finally the completely opened stethoscope was moved about from right to left below the thymus area, and the thymic area stroked in an oblique direction with a brush. This is especially valuable in the children over 1 year old. The results from this friction method were also noted.

The area of dulness thus mapped out was, as nearly as possible, estimated with reference to the sternum and ribs. It should be carefully noted how far from the median line of the sternum or the parasternal line the lateral borders come. The lower border should be measured with reference to the upper or lower edge of the second or third rib, and drawn in conformity to a diagram of the anterior thorax. Also, the greatest diameter of the dulness should be measured, vertically and horizontally.

The sketches made in this way of the topography and size of the thymus should be compared with the findings from an autopsy of the cases. In the cases we describe here, more than 140 were examined clinically, and one-tenth of these were confirmed by autopsy. We found in the cases examined considerable variations in the size of the thymus dulness. We were able, however, in place of these to use the material chosen for the physiologic data, in which we could find a large group of children of the same age among the well-developed and nourished ones.

It is already known that the thymus shows a constant development with the increasing age of the child, and grows until the age of puberty. Its growth is not uniform, however; its most intense growth is between the second and fifth years of life. It does indeed increase in weight until the fifteenth year, but in relation to the rest of the body is smaller. According to the estimates of v. Sury[20] and the careful examinations of Hammar[21] the thymus weight is reckoned:

	v. Sury gm.	Hammar gm.
In the new-born	14.4	13.26
From 1-5 years	22.1	22.98
From 6-10 years	29.4	26.10
From 11-15 years	32.2	37.50

While the thymus weight in the new-born bears a proportion of about 4-1,000 to the rest of the body mass, and in the first to the fifth year about 2.2-1,000, the proportion sinks by the tenth year to 1.2-1,000, and in the fifteenth year to 0.1-1,000.

After puberty a gradual shrinkage of the thymus sets in, a physiologic involution, while during exhausted periods at any age a marked reduction in size can be demonstrated, the so-called accidental involution. Soderlund and Backmann[22] in their animal experiments were able to show

20. Von Sury: Vierteljahrschr., f. gerichtl. Med., 1908.

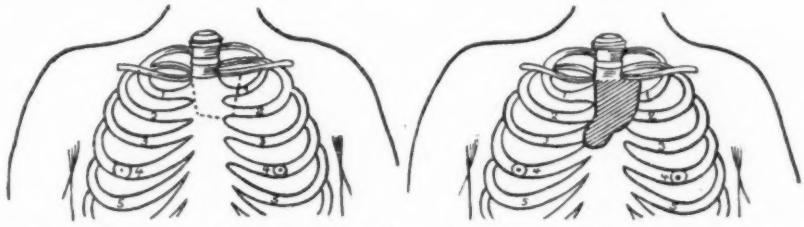
21. Hammar: Arch. f. Anat. u. Physiol., 1906; Vierteljahrsch. f. gerichtl. Med., 1908.

22. Soderlund and Backmann: Arch. f. microscop. Anat., 1909.

a reduction to one-tenth of the thymus weight after several days of starvation.

In general we concluded from the foregoing data that the greatest anatomic development and the most important biologic epoch could be looked for between the second six months and the third year of life, and therefore we selected this period for our physiologic experiments. In contrast to Blumenreich we found in this period a more rhomboid form of thymic dulness whose size and location varied with the age of the child. The lower edge generally reached to the second or third rib, and commonly ran in an oblique direction. And in agreement with Blumenreich and in contradiction of an old declaration of Sahli's, a clear full tone was demonstrable between the lower border of the thymus and the upper border of the heart.

While we try in the following experiments to give the most typical examples in each age classification, at the same time we will preferably regard only such cases as were proved by autopsy. In this way the worth of the physiologic experiments can be best judged. In children in the first half year of life we found the thymic dulness in the form of a small



Figs. 11 and 12.—Thymus findings in a child 4 years old which died of heart disease following diphtheria; size of thymus on dissection corresponded with percussion findings. A small, tongue-shaped lobe extended downward toward the right side, which could not be outlined by percussion.

trapezoid which stretched from the jugular to the second rib; the right border lay mostly in the right sternal line, but more often in the middle line. The left border generally extended over the right sternal line, but was not so sharply defined as the left.

In a case of a 4 months' child brought to autopsy three days after the original examination, the volume of the thymus at section overlapped the dulness area found clinically, as is shown in Figures 5 and 6, and this thymus lay deeper in the thorax than could possibly be ascertained physically.

In a child about 1 year old, with a chronic nutrition disturbance and subnormal body weight, in which the autopsy cited was done several months after the clinical findings were made, the two pictures are contrasted as in Figures 7 and 8. In a child a year and a half old, which had been in a very well-nourished condition and died of an intercurrent infection (descending croup), we outlined on the cadaver the thymus

dulness shown in Figure 9, and confirmed it by autopsy as seen in Figure 10. Here the dulness-area found covered the upper part of the thymus as seen at autopsy, but besides this a tongue-shaped lobe extended down toward the heart (Fig. 10), which lobe was so covered by a thick layer of lung tissue that to find it by percussion could not be expected.

In another case, confirmed at autopsy, a child about 4 years old of fairly good nutrition died of heart disease several weeks after a diphtheritic attack; the size of the thymus when dissected out was exactly that of the area of dulness found by percussion. Here there was also a small tongue-shaped lobe, but it extended toward the right side and forbade physical outlining (Figs. 11 and 12).

The thymus dulness of children between 2 and 3 years — a period we have so far been unable to study by autopsy — does not differ materially from the case of an 18 months child shown in Figure 9.

Thymus dulness may easily be wrongly diagnosed from other features in the mediastinal space.

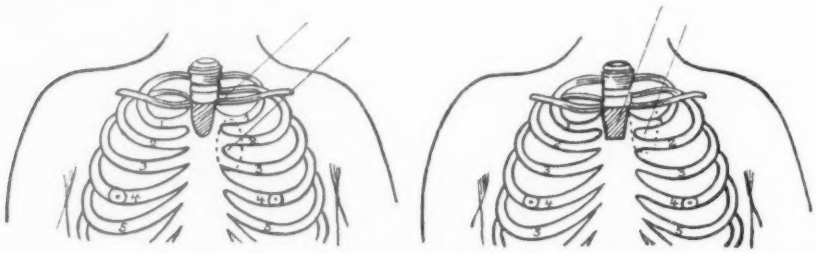


Fig. 13.—The area of atypic dulness outlined a caseous lymph-gland on the left side; thymus in the median line, small, and did not show perceptible dulness.

Fig. 14.—Atypical dulness caused by infiltration of left lung; thymus small and not demonstrable by percussion.

Of the factors which may be mistaken for enlargement of the thymus, the lymph-glands in childhood take first place, whether they are only swollen or are infected with tuberculosis. On the other hand such organs as can cause a dull area in the adult, like a struma or a mediastinal tumor, come under observation only exceptionally in the child, and may be ignored. Next in importance are the changes in the lung parenchyma, whether inflammatory or tuberculous, which, when they cause a dulness in the neighborhood of the thymus in children, cause deception in diagnosing thymus dulness.

In a part of the cases a conclusion may be reached that this dulness is not that of the thymus, because it lies in an atypical place, and does not show the median position of a thymus. Such atypical dulness should be regarded with distrust as not belonging to the thymus. If one finds foci of infiltration over the lungs, or a positive reaction for tuberculosis, one should be inclined to ascribe the dulness rather to the diseased foci. Auscultation will often decide this fact.

But if swollen or caseous lymph-glands lie under the thymus or are even adherent to it, it is almost impossible to differentiate these in the living. We have been able to draw some important conclusions from the autopsies of children we could examine during life, and show such cases in Figures 13, 14 and 15.

In Figure 13 the atypic dulness outlines a caseous lymph-gland on the left side, while the thymus lay in the median line, was very small, and did not show a perceptible dulness.

In Figure 14 a similar atypical dulness is that of an infiltrated left lung, and the thymus was here also very small and not demonstrable by percussion.

In Figure 15 again the caseous lymph-glands lie in the median line and are adherent to the thin thymus and present the picture of an enlarged thymus. Only the fact that a Pirquet reaction was positive in

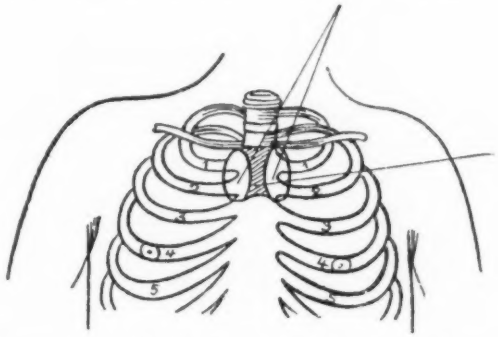


Fig. 15.—Caseous lymph-glands in the median line adherent to the thymus, presenting the picture of an enlarged thymus.

the seventh month, had warned us that we must not ascribe this dulness solely to the thymus.

Among the cases examined so far we found besides the common signs pertaining to the thymus, a certain multiplicity of facts which lead us to believe that we have made a step forward in the knowledge of the condition of the thymus in childhood, and shall make others. On account of the incompleteness of the physical methods, however, we must exert ourselves to find further functional reactions, which will help to broaden the results of physical examination.

CONCLUSIONS

Combining an exact percussion of the mediastinal area, which is done with the new percussion instrument, with auscultatory percussion and friction of this region, it was possible to obtain a more exact estimate of the size and condition of the thymus than has hitherto been the rule.

Systematic examination of 140 cases shows that the size of the thymus varies according to the age of the child and its nutritive condition, and to certain pathologic conditions.

Normally the area of thymus dulness in children shows a rhomboid form, which extends from the jugular down to the second or third rib, and whose lateral borders are confined to the sternal and parasternal lines. By exact drawings of the clinical findings on a diagram of the anterior thorax they can be correctly compared with the autopsy results.

A STUDY OF THE PYROGENIC ACTION OF LACTOSE*

F. W. SCHLUTZ, M.D.
Instructor in Pediatrics, University of Minnesota
MINNEAPOLIS

The question whether a food component if introduced into the intermediary metabolism unchanged is capable of developing pyrogenic action has received renewed interest since Finkelstein[1] first published his observations on alimentary intoxication.

Finkelstein designates this fever as alimentary fever because he believes it dependent on the pathologic effect of some food component introduced into a diseased intestinal tract.

Other observers have held similar views, instances of which are the efforts of Krehl and Matthes[2] and Schultes[3] to show this effect for albumoses; also the observations of Weill and Tiberius,[4] who called attention to a definite relationship existing between temperature variations and food given artificially-fed infants.

The so-called buttermilk fever of Tugendreich[5] has been regarded in the light of a sugar fever. Most observers agree that the food component develops pathologic activities only if there is primarily a lesion in the intestinal tract, an alteration in the wall, functional or otherwise, which permits the food component to pass the barrier and enter the intermediary metabolism not properly changed.

This alteration is brought about by inflammatory changes caused according to Bokai[6] by fatty acids, and as Finkelstein[1] and v. Reuss[7] believe and Jacoby[8] and Escherich[9] believed as long ago as 1877 and 1887;

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*From the Laboratory of Physiology, University of Minnesota. Director, Professor R. O. Beard.

*Read before the Chicago Pediatric Society.

1. Finkelstein, H.: Ueber alimentäre Intoxikation, Jahrb. f. Kinderh., lxv, 263.

2. Krehl, L., and Matthes, M.: Ueber die Wirkungen von Albumosen verschiedener Herkunft, Arch. f. Path. u. Pharmakol., xxxvi, 437. Matthes, M.: Ueber die Wirkung einiger subcutan einverleibten albumosen auf den tierischen Organismus, Deutsch. Arch. f. klin. Med., liv, 39.

3. Schultes: Die Beziehungen zwischen Albumosurie und Fieber, Arch. f. klin. Med., lviii, 325, and lx, 55.

4. Weill and Tiberius: La temperature dans les differentes formes d'allaitement chez les nourissons sains, Rev. mens. d. mal. de l'enf., xxi, 168.

5. Tugendreich: Ueber buttermilch Fieber, Arch. f. Kinderh., xliv, 21.

6. Bokai: Experimentelle Beiträge zur Kenntniss der Darmbewegungen, Arch. f. exper. Path. u. Pharmakol., 1888, xxiv, 153.

7. v. Reuss: Die Bedeutung der Kohlehydrate für die Ernährungs-Störungen im Säuglingsalter, Wien. med. Wchnschr. Nos. 28, 29 and 30, 1910.

8. Jacoby: Die Pflege u. Ernährung des Kindes, Gerhardt's Handbuch, 1877, p. 409; Milk Sugar in Infant Feeding, Arch. Pediat., 1901, xviii, 801.

9. Escherich: Verhandl. der 5ten Gesellsch. f. Kinderh., Wiesbaden, 1887, 31.

also by lactose or acids developed through the fermentation of the sugar.

That lactose particularly possesses this effect has been abundantly demonstrated both experimentally and clinically, and this has been explained on the basis of experiments by Albertoni[10] and Röhman and Nagano[11] showing that lactose is more slowly absorbed from the intestinal tract than other sugars. Affected by some change or inflammation, the intestinal wall becomes permeable for substances which otherwise it does not permit to pass unchanged.

Meyerhofer and Pribram[12] were indeed able to show increased resorption and heightened permeability of the intestinal wall for such substances as sugar. Similar effects were observed by Cohnheim,[13] who found that toxic substances introduced into the intestinal tract could alter its permeability.

Langstein and Steinitz[14] came to similar conclusions, as did also Ganghofer and Langer.[15]

The fate of lactose when introduced into the human or animal organism has been the subject of much experiment. Most of it directed primarily toward the determination of the tolerance of the organism for lactose and then secondarily toward the factors which influence this tolerance in health and disease.

F. Voit,[16] v. Halasz,[17] Cremer,[18] Blumenthal,[19] Hofmeister[20] and Helmholz[21] found that the animal organism's tolerance for lactose is

10. Albertoni: Sal contegno e sull'azione degli zuccheri nell' organismo, Ann. di chim. et di Farm. Milano, 1891, xiii, 145.

11. Röhmann and Nagano: Ueber die Resorption und die fermentative Spaltung der Dissacharide im Dünndarm des ausgewachsenen Hundes, Arch. f. d. ges. Physiol. (Pflüger), xcv, 533.

12. Meyerhofer and Pribram: Das Verhalten der Darmwand als osmotische Membran bei akuter u. chronischer Enteritis, Wien. klin. Wchnschr., 1909, xxii, 875.

13. Cohnheim: Die Resorption im Dünndarm und in der Bauchhöle, Ztschr. f. Biol., xxxvii, 443.

14. Langstein and Steinitz: Laktase und Zuckerausscheidung beim magendarm kranken Säuglinge, Beitr. z. chem. Phys. u. Path. (Hofmeister), vii, 515.

15. Ganghofer and Langer: Ueber die Resorption genuiner Eiweisskörper im Magendarmkanal neugeborener Tiere und Säuglinge, München. med. Wchnschr., 1904, l, 1497.

16. Voit, F.: Untersuchungen über das Verhalten verschiedener Zuckerarten im menschlichen Organismus nach subcutaner Injection, Deutsch. Arch. f. klin. Med., lviii, 523.

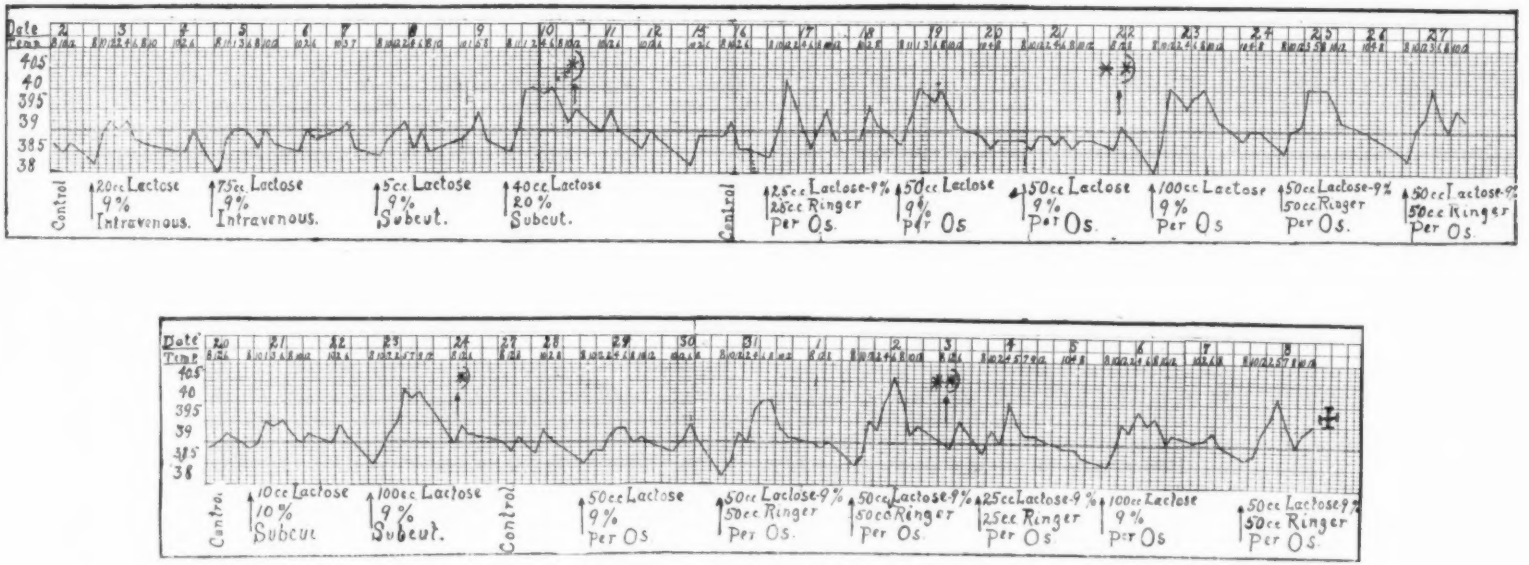
17. v. Halasz: Die Resorption und das biologische Verhalten der verschiedenen Zuckerarten im Dickdarm, Deutsch. Arch. f. klin. Med., 1910, xcviii, 433.

18. Cremer, M.: Zucker und Zelle, Ztschr. f. Biol., xxxii, 49.

19. Blumenthal: Zur Lehre von der Assimilationsgrenze der Zuckerarten, Beitr. z. chem. Phys. u. Path. (Hofmeister), vi, 329.

20. Hofmeister: Ueber Resorption und Assimilation der Nährstoffe, Arch. f. exper. Path. u. Pharmakol., xxv, 240.

21. Helmholz: Studies on Milk-Sugar, Arch. Pediat., xxviii, 373.



Charts 1 and 2.—Temperature curves resulting from the giving of lactose and lactose and Ringer's solution to rabbits with intestinal disturbance produced by feeding beta-oxybutyric acid and croton oil. The star in each chart indicates the point at which butyric acid feeding was begun; the double star the point at which croton oil feeding was begun.

lower than for any other sugar. This tolerance sinks particularly low in diseases of the intestinal tract, as was shown by experiments of Grosz,[22] Koplik,[23] Nobécourt[24] and Aschenheim,[25] and more recently by v. Reuss,[26] Leopold[27] and Nothmann.[28]

This led to considerable study of the enzyme lactase, particularly the bearing its presence or absence had on the occurrence of lactosuria. It is present early in life. Miura[29] found it in the intestine of the still-born and Ibrahim[30] and Nothmann[31] in premature infants. Weinland[32] and Orban[33] always found it present in the intestine of the healthy infant but absent in disease. That it is also present in the diseased intestinal tract though in diminished amount was demonstrated by Langstein and Steinitz.[14]

These results and the definite clinical evidence that there seemed close connection between lactose and the production of the pronounced symptoms of alimentary intoxication, principally the fever, led Finkelstein to look on lactose as a substance possessing pyrogenic properties.

The experiments of L. F. Meyer,[34] since confirmed by Friberger,[35]

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22. Grosz, J.: Beobachtungen über Glycosurie im Säuglingsalter nebst Versuchen über alimentäre Glycosurie, Jahrb. f. Kinderh., xxxiv, 83.

23. Koplik: Dietetic Glycosuria in Artificially Fed Infants, with Cases, Arch. Pediat., 1892, ix, 781.

24. Nobécourt: De l'elimination par les urines de quelques sucres introduits par la voie digestive ou la voie sous-cutanée chez les enfants, Rev. mens. d. mal. de l'enf., xviii, 161.

25. Aschenheim: Zuckerausscheidung im Säuglingsalter, Verhandl. d. Gesellsch. f. Kinderh., xxvi, 178.

26. v. Reuss: Ueber alimentäre Sacharosurie bei darmkranken Säuglingen, Wien. klin. Wchnschr., 1910, xxiii, No. 4, 123.

27. Leopold: Ueber die Wirkung der verschiedenen Zuckerarten bei ernährungsgestörten Säuglingen, Ztschr. f. Kinderh., i, 217.

28. Nothmann, H.: Beiträge zur Zuckerausscheidung im Säuglingsalter, ii, 503.

29. Miura: Ist der Dünndarm im Stande Rohrzucker zu invertieren, Ztschr. f. Biol., xxxii, 266.

30. Ibrahim, J.: Zur Verdauungsphysiologie des menschlichen Neugeborenen, Ztschr. f. physiol. Chem., Strassb., 1910, lxiv, 95; Ibrahim, J., and Kaumheimer, L.: Die Doppelzuckerfermente (Laktase, Maltase, Invertin) beim menschlichen Neugeborenen und Embryo, Ztschr. f. physiol. Chem., Strassb., 1910, lxvi, 19.

31. Nothmann, H.: Laktase- und Zuckerausscheidung bei Frühgeborenen Säuglingen, Monatschr. f. Kinderh., viii, 377.

32. Weinland, E.: Beiträge zur Frage nach dem Verhalten des Milchzuckers, Ztschr. f. Biol., xxxviii, 16.

33. Orban, R.: Ueber das Vorkommen der Laktase im Dünndarm und in den Säuglingsfæces, Prag. med. Wchnschr., 1899, No. 33; cited from Maly's Jahresb. d. Tierchemie, xxix, 384.

34. Meyer, L. F.: Die Bedeutung der mineral Salze bei den Ernährungsstörungen des Säuglings, Verhandl. der Gesellsch. f. Kinderh., Salzburg, xxvi, 1; Experimentelle Untersuchungen zum alimentären Fieber, Deutsch. med. Wchnschr., 1909, xxxv, 194.

35. Friberger: Untersuchungen über das sogenannte Salzfieber, München. med. Wchnschr., 1909, lxi, 1946.

Schloss,[36] Cobliner[37] and Nothmann,[38] demonstrating conclusively that the Na-halogen group of salts possessed pyrogenic activity, brought out a new factor and caused Finkelstein[39] to regard the pyrogenic action of the sugar as possibly a salt action.

Finkelstein considered that the fever was brought about through some physical action of these substances, essential to which was a preliminary breaking down of the apparatus which maintained the constancy of the tissue fluids. Through this breaking down the salt or the body analogous to it, in this case lactose, entered the circulation in such large amounts that the physiologic state of concentration was altered, and this alteration resulted in fever. There was another possibility. The breaking down of the barrier caused an alteration of vital factors which under normal condition would prevent pyrogenic action of these substances.

From a recent communication it appears that Finkelstein,[40] influenced no doubt by newer investigations, has modified his theory in regard to the pyrogenic effect of lactose in alimentary intoxication to this extent: He believes that under natural conditions (per os) so much is removed by fermentation and resorption that the physical effect (pyrogenic action — analogous to salt action) does not take place. The sugar and the acids developed from it through fermentation largely confine their effect to the production of intestinal lesions (possibly functional) which in turn prevent the proper metabolism of the salts and permit these to develop their physical effect.

Finkelstein's theory that the action of lactose was probably a physical action seemed to be supported by experiments carried on by Schaps[41] in 1907 at the *Kinder-Asyl*.

Schaps observed typical temperature rises after subcutaneous injections of isotonic solutions of NaCl, as well as solutions of dextrose and lactose. The fever reached its fastigium after eight to ten hours and disappeared after twenty-four hours. Only osmotically equal solutions of mono- and dissaccharids seemed to possess pyrogenic action. This fact led Schaps to conclude that the action of sugar was analogous to a salt action, a view which Keller[42] had at one time expressed, and that the pyrogenic effect was brought about in the following manner: Salt or the substance physically analogous to it, in this case lactose, acts pyrogenically when it enters the circulation, either directly, as a substance pos-

36. Schloss, E.: Studien über Salzfieber, Biochem. Ztschr., 1909, xviii, 14.

37. Cobliner, S.: Beiträge zum Kochsalzfieber, Ztschr. f. Kinderh., ii, 439.

38. Nothmann, H.: Zur Frage des Kochsalzfiebers beim Säugling, Ztsch. f. Kinderh., i, 73.

39. Finkelstein: Ueber alimentäre Intoxication, Jahrb. f. Kinderh., lxviii, 521 and 692.

40. Finkelstein: Personal Communication.

41. Schaps, L.: Salz und Zucker injectionen beim Säugling, Berl. klin. Wchnschr., 1908, xlv, 1309.

42. Keller, A., Czerny, A., and Keller, A.: Des Kindes Ernährung, etc., ed. 1, Leipzig, F. Deuticke, i, 326.

sessing pyrogenic action, or by virtue of an alteration in the molecular concentration which its presence in the tissue fluids occasions.

Similar experiments with subcutaneous injections of lactose were carried on in infants and animals by Leopold and v. Reuss.[43] Their results coincided fully with those of Schaps and both arrived at similar conclusions. Leopold has also very recently published some observations on lactose and its connection with temperature rises in alimentary disturbances which seem to coincide fully with Finkelstein's later views as expressed above.

Contrary to these results are some observations which do not coincide with such rises as expressed by Finkelstein or Schaps.

Nobécourt[24] and Keller[44] both injected concentrated solutions of lactose subcutaneously to determine the tolerance for it. Outside of severe local reactions neither author saw untoward toxic effect from these injections and makes no mention of fever. J. v. Kossa[45] reports similar effects. This has raised the doubt in some minds as to whether the temperature recorded by Schaps was not entirely due to local reaction caused by tissue injury at the seat of injection.

Neither F. Voit[16] nor Dastré[46] record any fever reactions with the intravenous administration of lactose. The latter's experiments are particularly interesting since he gave the lactose in physiologic salt solution.

In all the experiments of Gross,[22] Nobécourt,[24] Terrien[47] and Keller,[44] dealing with alimentary glycosuria and lactosuria and the determination of the tolerance the human and animal organism has for the various sugars, no mention is made of pyrogenic effect.

Cobliner[48] has recently published the interesting observation that in dyspepsias and alimentary intoxication the sugar content of the blood is not increased.

Rosenthal[49] in an extensive series of experiments on dogs and rabbits was able to show pyrogenic effect for lactose in only a few instances. With salt solution he found even hypothermia and concluded that neither one possessed pyrogenic property or was the principal factor in the production of alimentary fever.

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43. Leopold and v. Reuss: Experimentelle Untersuchungen über Milchzuckerausscheidung nach wiederholten subcutanen Injectionen, Monatschr. f. Kinderh., viii, 1 and 453.

44. Keller, A., Czerny, A., and Keller, A.: Des Kindes Ernährung, etc., i, 320.

45. Kossa, J. v.: Beitrag zur Wirkung der Zuckerarten, Arch. f. d. ges. Physiol. (Pflüger), lxxv, 310.

46. Dastre: Observations on Diuresis Produced by Various Sugars, Compt. rend. Soc. de biol., xli, 574.

47. Terrien: De la glycosurie alimentaire chez les nourissons, Rev. mens. d. mal. de l'enf., xviii, 402.

48. Cobliner, S.: Blutzucker Untersuchungen bei Säuglingen, Ztschr. f. Kinderh., i, 207.

49. Rosenthal, F.: Zur Frage des alimentären Fiebers, Jahrb. f. Kinderh., lxx, 123.

The experiments of Dastré[46] and F. Voit[16] are the only ones recorded in the literature in which lactose was given intravenously.

At Professor Finkelstein's suggestion I undertook this study which primarily had as its object the observation of the effect of lactose on the body temperature if introduced directly into the blood-stream in varying amounts and under varying conditions. I eventually enlarged on this in order to study the general effect of lactose and compare my results with those of other observers. I selected the rabbit for experimentation on account of the ease with which intravenous infusion and all other manipulations can be carried out on this animal. The technic of the experiment was as follows:

Female rabbits were generally selected and were liberally fed on carrots and other vegetables. The temperature of the rabbit was controlled at regular intervals the day before the experiment. The highest temperature on this day was considered as the control. Any rise above it was considered as a febrile reaction.

The temperatures were taken at regular intervals of from one-half to three hours. The thermometer was introduced about 4 to 5 cm. into the rectum and allowed to remain five minutes. The normal temperature of the rabbit, while somewhat labile, seems to lie between 38.5 and 39.5 C. These figures coincide with extensive observations made on this point by Gibson.[50]

The intravenous and subcutaneous administrations were carried out under absolute aseptic precautions and only sterilized solutions used in all the experiments. The solutions were introduced into a vein of the ear, under the skin of the abdomen, and orally by means of a stomach tube.

In the series of experiments with butyric acid and croton oil feeding, these substances were given orally in watery solution 0.5 per cent. strength and in olive oil 10 minims to 20 c.c., respectively. Eighty experiments were carried out covering in all eight different series.

Five intravenous injections with distilled water in amounts varying from 10 to 75 c.c. were followed three times by febrile reactions, coinciding in this respect with similar experiments of Helmholz,[51] who believes that this action is due to the destruction of blood cells and the setting free of pyrogenic substances.

Lactose alone was given intravenously twenty-three times in concentration of 5 per cent. up to 50 per cent., and amounts varying from 6 to 50 c.c. There were nineteen positive reactions. The time to the fastigium varied between two and seven and one-half hours. The general rise of temperature for all experiments was 0.36 degree. The highest

50. Gibson: On Proteose Fever, Doctorate Thesis, Yale, 1906.

51. Helmholz: Pyrogenic Action of Salt Solutions in Rabbits, Arch. Int. Med., vii, 468.

temperature recorded was 1.3 degrees above the control. This animal died five hours after the injection. It had some convulsions. The autopsy was negative.

TABLE 1.—INTRAVENOUS INJECTIONS

Number of Experiments	Conc. in Per Cent.	Quantity Used, c.c.	Time to Fastigium, Hours	Highest Temperature, C.	Rise Above Control, Degrees	Animal*
EXPERIMENTS IN WHICH DISTILLED WATER WAS USED						
1	..	10	...	39.1	0.0	A
6a	..	10	2	40.0	0.6	B
7a	..	20	4	39.7	0.1	B
2	..	50	2	39.8	0.5	A
3	..	75	...	38.6	—0.2	A
				Average	0.25	
EXPERIMENTS IN WHICH LACTOSE WAS USED						
1a	5	6	...	38.8	—0.4	B
2a	5	10	7½	40.	0.6	B
3a	5	10	3½	39.8	0.6	B
6	5	20	5	40.	0.5	A
7	5	40	...	39.	—0.2	A
8	9	10	4	39.9	0.2	A
9	9	10	3½	40.0	0.6	A
9a	9	20	6	39.8	0.8	B
10	9	20	4	39.2	0.15	B
10a	9	75	0	39.0	0.0	B
11	10	10	2	40.0	0.4	A
12	10	20	4	39.6	0.1	A
13	10	20	...	39.	—0.1	A
14	10	50	5	40.6	0.8	A
15	10	75	6	40.1	1.1	A
16	10	75	...	39.4	0.1	A
3a	10	100	4	39.9	0.3	B
2a	10	100	5	39.8	0.2	B
11a	20	20	...	39.	0.1	B
12a	20	50	...	39.3	0.1	B
13a	40	60	5	39.2	.00	B
16a	50	50	4	40.6	1.3	B
8a	50	50	5	39.6	0.8	B
				Average	0.36	

* A, unused animal; B, used animal.

In two further series I tried combinations of physiological salt solution and lactose, and Ringer's solution and lactose. The lactose was given in solutions of 9 and 10 per cent., respectively. There was a positive rise of temperature above the control in every instance. The average rise for the series with physiological salt solution and lactose was 0.71 degree and for the series with Ringer's solution 0.9 degree. The time to the fastigium was from two to six hours. The highest rise above the control, 41.6 C., occurred in an unused animal and was the highest temperature observed in any experiment. Whether a used or unused animal was taken for injection seemed to have no effect on the temperature rise.

TABLE 2.—INTRAVENOUS

Number of Experiments	Conc. in Per Cent.	Quantity Used, c.c.	Time to Fastigium, Hours	Highest Temperature, C.	Rise Above Control, Degrees	Animal*
		LACTOSE SOL., PHYSIOL. NaCl SOL. EQUAL PARTS				
25	10 NaCl	20	2	40.8	1.0	A
26	10 NaCl	10	2	40.2	0.8	A
27	10 NaCl	20	2	39.9	0.2	A
29	10 NaCl	10	2	40.8	0.9	A
30	10 NaCl	50	2	40.6	.85	A
					Average 0.71	
		LACTOSE AND RINGER, EQUAL PARTS				
32	10 R	20	4	41.6	2.0	A
31	10 R	10	3½	40.8	1.6	A
27	10 R	20	4	39.9	0.6	B
28	10 R	10	5	39.8	0.4	A
31a	10 R	50	4	40.	0.8	B
30a	10 R	50	4½	39.9	1.0	B
28a	9 R	30	5	40.	0.6	B
26a	9 R	30	4	39.3	0.3	A
					Average 0.90	

* A, unused animal; B, used animal.

TABLE 3.—SUBCUTANEOUS INJECTIONS

Number of Experiments	Conc. in Per Cent.	Quantity Used, c.c.	Time to Fastigium, Hours	Highest Temperature, C.	Rise Above Control, Degrees	Animal*
		LACTOSE USED				
1b	9	20	...	38.8	0.00	B
2b	9	10	4	39.6	0.1	B
3b	9	10	...	39.	—0.2	B
11b	9	20	5	39.9	0.4	B
12b	9	50	3	39.2	—0.1	B
9b	9	50	4	40.0	0.8	B
24a	9	100	7	40.2	1.0	B
22a	9	100	9	40.3	0.9	B
24	9	5	...	39.	—0.1	A
10b	9	5	...	39.2	0.0	B
22	10	10	5	39.5	0.3	A
10c	20	40	9	40.0	0.6	B
					Average 0.34	

* A, unused animal; B, used animal.

Twelve injections subcutaneously given, to my surprise, gave the lowest general average rise of temperature of all the experiments. Only seven animals reacted positively while three failed by 0.1 and 0.2 degree to reach the highest control temperature. The amounts injected varied from 5 to 100 c.c. The concentration of the lactose solution used was 9 per cent., 10 per cent., and one injection of a 20 per cent. solution. Neither the amount of fluid used nor the concentration had any appreciable effect on the temperature. Two animals received successive injections at intervals of a few days. In one the amount injected the first time was 5 c.c. and the concentration 9 per cent. No reaction ensued.

In the second injection the amount was 40 c.c. and the concentration 20 per cent. The reaction following was only 0.6 per cent. higher than the control the day before.

Similarly in the second animal the amounts were 10 and 100 c.c., respectively, followed by reactions of 0.3 and 0.9 degree. None of these animals, with the exception of the one which received the 40 c.c. of a 20 per cent. concentration, showed any pronounced local irritation at the seat of injection. The results are distinctly negative and do not coincide with the findings of either Schaps or Leopold and v. Reuss.

TABLE 4.—ORAL ADMINISTRATION

Number of Experiments	Conc. in Per Cent.	Quantity Used, c.c.	Time to Fastigium, Hours	Highest Temperature, C.	Rise Above Control, Degrees	Animal*
			LACTOSE USED			
3c	5	50	...	38.8	0.0	B
4	5	100	5	39.4	0.05	A
5	9	20	...	39.0	—0.1	A
6b	9	20	4	39.3	0.2	B
7b	9	50	...	38.6	—0.4	B
13b	9	100	8	40.0	0.3	B
14b	10	50	...	39.1	0.0	B
15a	20	50	...	38.6	—0.4	B
8b	50	50	6½	40.1	0.8	
				Average	0.15	
			LACTOSE AND SODIUM CHLORID SOLUTION			
4a	9 NaCl	50	6	39.9	0.3	B
5a	9 NaCl	100	3	39.	0.0	B
17	9 NaCl	30	8	39.6	0.5	A
18	10 NaCl	50	8½	40.0	0.6	A
				Average	0.35	
			LACTOSE AND RINGER'S SOLUTION			
19	9 R	30	6½	39.5	0.5	A
10d	9 R	50	9	39.3	0.1	B
20	9 R	100	6	40.1	0.9	A
				Average	0.50	

* A, unused animal; B, used animal.

Finally I gave lactose both alone and in combination with physiological salt solution orally. First in a series of healthy rabbits, that is, rabbits free from any apparent alimentary disturbance, and then in a series of rabbits in which more or less pronounced intestinal disturbance had been produced by feeding beta-oxybutyric acid and croton oil, respectively (Charts 1 and 2).

Lactose given alone in amounts varying between 50 and 100 c.c. and in concentration of from 5 to 50 per cent. only once produced an appreciable rise of temperature over the control. This was 0.8 degree, follow-

ing the administration of 50 c.c. of a 50 per cent. solution. In nine experiments only four reactions were shown above the control.

The general average rise for lactose and physiological salt solution and lactose and Ringer was only 0.35 and 0.50 degree, respectively, very little higher than the reactions with lactose alone.

TABLE 5.—ORAL ADMINISTRATION, BETA-OXYBUTYRIC ACID AND CROTON OIL FEEDINGS

Number of Experiments	Conc. in Per Cent.	Quantity Used, c.c.	Time to Fastigium, Hours	Highest Temperature, C.	Rise Above Control, Degrees	Animal*
		LACTOSE AND RINGER'S SOLUTION				
10e	9 R	50	4½	40.2	1.0	B
10f	9	50	5	40.	0.6	B
10g	9	50	...	38.9	—0.1	B
10h	9	100	4	40.	0.9	B
10i	9 R	100	9	40.	1.0	B
10j	9 R	100	7½	40.1	1.1	B
23	20 R	50	6	40.	0.8	A
22b	9	50	8	39.8	0.5	B
22c	9 R	100	10	40.	0.6	B
22d	9 R	100	10	40.6	1.6	B
22e	9 R	50	9½	40.	0.5	B
22f	9	100	8	39.8	0.9	B
22g	9 R	100	9	40.1	0.8	B
33	9 R	100	7	41.	1.0	B
33a	9 R	50	...	39.2	—0.3	B
				Average	0.76	
		LACTOSE AND SODIUM CHLORID SOLUTION				
33b	9 NaCl	100	7½	40.	0.2	B

* A, unused animal; B, used animal.

The last series in which the rabbits were fed beta-oxybutyric acid and croton oil in order to produce intestinal lesions, proved the most interesting. That this disturbance is quite readily accomplished is evidenced by the diarrhea following the feedings, occurring usually after the fourth day in the oxybutyric acid feeding and somewhat earlier with croton oil. Several autopsies performed on animals just at the onset of the diarrhea showed more or less pronounced inflammatory changes in the intestinal wall. Animals killed at a later stage, in which the feeding had extended over a period of six or seven days, showed still more pronounced inflammatory changes.

The administration of the sugar and combined sugar and salt solutions was begun with the onset of the diarrhea and in several experiments continued at intervals of one and two days. In five of the experiments lactose alone was given in amounts varying from 50 to 100 c.c. and in 9 per cent. concentration. The highest reaction above the control following the giving of 100 c.c. of the solution was 0.9 degree. This same animal in two preceding experiments in which only 50 c.c. of a 9 per

cent. solution were given reacted with a rise of 0.6 degree the first time and failed by 0.1 to reach the control the second time. Subsequently it was twice given 100 c.c. of a combination of lactose 9 per cent. and Ringer's solution, equal parts. The highest reaction in the first experiment was 40, in the second 40.1 C. This was 1 and 1.1 degrees higher than the control. The animal at the time had profuse diarrhea, but showed no pronounced evidence of intoxication.

Lactose 9 per cent. and Ringer's solution combined were given in six other experiments in amounts varying from 50 to 100 c.c. The highest temperature recorded was 41 C. following the administration of 100 c.c. This same animal showed no reaction a few days later with 50 c.c. solution of the same concentration and only a rise of 0.2 degree a day later after the giving of 100 c.c. of equal parts of 9 per cent. lactose and physiologic salt solution.

This animal was given lactose in 9 per cent. concentration alone in one experiment and then in three successive experiments was given equal parts of lactose 9 per cent. and Ringer's solution. The animal at the time had profuse diarrhea and died the day following the last experiment. The temperature rose 1.6 degrees above the control only once in the four experiments. The charts of Animals 10 and 22 are appended.

Reviewing the series as a whole, the reaction was positive in fourteen out of sixteen experiments. Individually also the rises above the control were higher than in any of the other experiments, reaching or exceeding in five instances 1 C. My results are at variance with those of Schaps[41] and Leopold and v. Reuss[43] and coincide in the main with those of Rosenthal.[49]

CONCLUSIONS

The results of my experiments warrant the following conclusions:

1. Lactose if given intravenously, subcutaneously or orally, possesses no distinct pyrogenic effect no matter in what concentration or amount it is given.

2. It does possess a definite though not pronounced influence on the temperature if it is given subcutaneously or orally in an animal with a diseased intestinal tract in combination with a medium containing a sodium salt, such as physiologic or Ringer's salt solution.

These results though definite are hardly so to the extent expected when one considers the pronounced effect produced clinically by the administration of lactose in similar conditions, and are far from explaining the pathogenesis of the fever occurring in alimentary intoxication.

820 Donaldson Building.

THE MANAGEMENT OF SQUINT IN CHILDREN

C. W. LeFEVER, M.D.
PHILADELPHIA

It is of the greatest importance that those who preside over the physical destinies of children should understand that squint in children is curable. By squint I mean the common form of concomitant convergent strabismus, or "cross-eyes." There are other forms of squint occurring in children which are not curable except through operative measures, but they are comparatively rare.

When I state that the common squint is curable, I mean that it may be cured by non-operative measures, but the pediatrist must understand that the cure depends on the *early* institution of the proper measures and their implicit observance. The responsibility for allowing children to become adults with eyes crossed and loss of function in one eye — an eye otherwise normal — must be shared by three persons, whom I name in the order of their importance.

First, the parents. Many parents have a false pride which leads them into the error that it is more of a stigma to the child to wear glasses than it is to grow up with eyes crossed. (It must be said in justification of them that they are not usually aware that there will also be loss of function in one eye.) Some mothers are too much occupied to possibly give the child such attention as is necessary to carry out the treatment properly. Some parents are too poor to afford the glasses and the necessary repairs. And then there are some too careless or stupid to do their duty by their offspring.

Second, the pediatrist or general practitioner. Nearly every child is taken to a physician for advice when the squint first becomes manifest. Then it is that great stress should be laid on prompt action and the significance of both treatment and neglect should be made clear to the parents. It is not enough to tell the parents that the child should be taken to an oculist. They must be told when and where the oculist can be found and must be made to understand that an optician, by whatever name he may call himself, is not the proper person. Then, to the shame of the medical profession, there are still a few physicians who tell the parents not to be alarmed, that children often "outgrow" squint, and if they do not "it can be straightened when the child is old enough." The family physician is most certain to know of a squint whether he is consulted concerning it or not and it is his business to make inquiries as to what is being done, if anything, for its cure. Many well-meaning

parents do not know the difference between an optician and an oculist and may allow the years during which the child might be cured, to lapse by placing the child in the wrong hands.

Third, the oculist. Once the child reaches the oculist the responsibility rests with him, not only for the management of the squint, but that of the child and its parents as well. If a child is brought very young to the oculist, say at 1 year of age, he should congratulate both himself and the parents, and not tell the parents to bring the child back "when it is old enough to glass." This was formerly a common practice among oculists and is unfortunately still the custom with some.

I believe 100 per cent. of cures are possible, in the class of cases referred to, under the most favorable management, if the children are seen early enough.

It is of importance that every physician should know about the amblyopia; should know that nearly all of these children lose useful vision in one eye, and that the restoration of its function is possible, and a necessary part of the treatment.

Before outlining the individual management of the patient, it is necessary to state the essentials of the etiology. Two factors are responsible for the squint. They may act together or singly.

1. Nearly all such children are far-sighted. Either the cornea is too flat or the scleral axis is too short, so that the rays of light entering the eye are not yet focussed when they reach the retina. This causes the object looked at to be seen indistinctly unless the patient can add to his refraction, by means of the accommodation, sufficient to cause the rays to meet in the macula. This he can usually do and the intuitive desire for clear vision leads him to do so. He accomplishes this by excess stimulus over the third nerve which supplies the muscle of accommodation. Now, since the third nerve also supplies stimulus to the converging group of muscles, and the stimuli for accommodation and convergence are synchronous, an excess also goes to the convergers. When there is no error of refraction neither accommodation nor convergence is required for distance fixation, and for nearer points the amount of stimulus for each corresponds, unit for unit, with the other. But when the patient is far-sighted, stimulus is necessary to the accommodation for distance fixation where no convergence is required, and at nearer points the accommodation stimulus is always in excess of that required for convergence. Since clear vision is the chief function of the eye the accommodation element of the stimulus predominates and too much stimulus is given to the convergence. This would cause the optic axes of the two eyes to cross and a squint to occur in all hyperopes were there not some means by which the patient can compensate for the excess convergence. This he does by sending as much excess stimulus over the sixth nerve to the external rectus muscle as there

is excess convergence, and the eye is drawn tightly back into its suspensory ligament, giving the deep-set eye, characteristic of hyperopia. Or the patient may be able to separate the two stimuli and converge less than he accommodates.

Most hyperopes succeed, in one of these ways, in keeping the optic axes parallel or properly converged, but some fail and squint is the result.

2. The fusion sense presides over binocular single vision, recognizing any failure of the two images to occupy identical points in the two maculas. It is readily understood that no mechanical adjustment could accomplish single binocular vision, when it is known that the size of the image on the retina may not exceed 1/50 mm. (1/1250 of an inch.) The fusion sense, acting through the coordination center, is therefore necessary to accomplish this wonderful physiologic feat. Now, the fusion sense, while hereditary, is undeveloped at birth, just as other functions are, and in some children its development is slow or may be arrested. In such a case there is deficient effort made at compensating for the overstimulus of the convergence in hyperopia and the eyes cross.

If the child happens to be near-sighted there is not sufficient third nerve stimulus and the squint is apt to be divergent. But myopia is more often acquired than congenital, so that the divergent type is not often seen in young children.

TREATMENT

The treatment is best considered under the following five heads:

1. Correction of the hyperopia removes the chief cause of the squint and is therefore of prime importance. At what age may a child be glassed? There is no definite answer to this question, but instances are very rare in which the eyes become permanently crossed in a child too young to wear glasses, if it can have proper supervision. Rarely is the patient seen before the age of 1 year, because the visual function is not sufficiently established before this age to call the accommodation into use for sharp vision. Young children take to glasses as they do to shoes or any other wearing apparel and give little trouble after the first week. It is my custom to glass them when first seen unless I am convinced that the glasses will not be worn, either from pride or lack of attention on the part of the parents. A former objection to early glassing was the difficulty in knowing what to order while the child was too young to read, but retinoscopy has eliminated this difficulty.

2. Atropin may be used to suppress the accommodation and render third nerve stimulus of no avail in helping the visual acuity, and the patient will soon cease the effort, incidentally ceasing to overstimulate convergence. This method, once much employed, should only be used when glasses are out of the question, and until such a time as the child may be glassed. It must be used in both eyes two or three times daily.

3. Cure of the amblyopia is essential if single binocular vision is ever to be established. Amblyopia is a form of partial blindness, of cerebral location. It comes about as the result of suppression of the image stimulus from one eye, in an effort to be free from diplopia. All these little patients doubtless see double at first, but are too young to explain the diplopia or even to know that it is unusual. As they learn that one object is false, and it perhaps leads them to make mistakes in taking hold of objects, they begin to ignore the dimmer one — and one is always dim because focussed on the peripheral retina where acuity is low. This suppression soon becomes permanent and some effort is required to give the child diplopia even with a small light and colored glass.

The squinting eye is now unable to see well even with its fellow covered, and is said to be amblyopic. The degree of amblyopia depends somewhat on the duration of the squint. Its cure depends on the forced use of the squinting eye, by occlusion of the good eye. Occlusion may be accomplished by a bandage worn permanently, for six weeks to two years, but since this is objectionable a better plan is to have a highly concave, neutral, frosted lens fitted closely over the good eye, while the proper correction is worn on the amblyopic eye. The obscured lens must rest against the side of the nose, fit closely around the rim of the orbit, and be turned back somewhat at the temporal end. This leaves room for the lashes without allowing the child to see around it. It must be worn constantly. This is not difficult to attain in very young children, say under the age of 5 years, but requires close watching for a month or so in older ones. The child should be kept in the room with its mother, if necessary tied to her apron-strings, until thoroughly accustomed to the use of the amblyopic eye. It is sometimes necessary to use atropin in the covered eye as an aid to prevent peeping around the glass.

I use this method as a routine and find it always successful in patients seen before they are too old. It is difficult of application after the seventh or eighth year. The cover should not be removed until the visual acuity is 20/30 or better. Both eyes are then glassed with the proper correction.

4. The fusion sense must be reestablished. If it were not originally at fault it becomes eliminated, and binocular single vision is impossible without it. In many cases it requires no treatment, resuming its function when the images can be fused. When the squint is of long standing, fusion may not be resumed without encouragement. For this purpose there are several instruments, all based on the principle of stereoscopic vision. Companion pictures are used and are either moved until they fuse or prism aid is given until fusion takes place while the child watches the pictures. The child is thus encouraged to see the same object with both eyes at the same time, without diplopia; i. e., single binocular vision is reestablished.

5. Operative measures are to be used only as a last resort and will very rarely be necessary if the child is properly managed from the incipiency of the squint. In patients not seen early or not properly managed, it becomes necessary to "straighten" the eyes in a mechanical way. This is done by shortening the external rectus, one or both, and tenotomizing the internus. The former is to be preferred to the latter and in any case full correction is to be avoided, as the future tendency is outward and a divergent squint may result in adult life. The operation should not "straighten" but aid other measures to do so. No patient should be operated on until the correction has been worn at least one or two years. The technic of the operative measures is not of interest here.

1708 Pine Street.

PROGRESS IN PEDIATRICS

RECENT ADVANCES IN OUR KNOWLEDGE OF RACHITIS

FRITZ B. TALBOT, M.D.
BOSTON

ETIOLOGY

Schmorl[1] gives a very extended review of the subject and I shall use his work freely without further reference to it. The following hypotheses have been advanced and are supported by more or less pathologic-anatomic data:

Artificial rachitis in animals: Most of the authors who have undertaken these experiments believe that rachitis is a disturbance of the calcium metabolism, and that it results either from giving too little calcium in the food, or that the normal calcium in the food is not absorbed in sufficient amounts, or finally, that it is absorbed and the bones are not able to assimilate it in a normal manner. The experiments have led to varying results. In most instances feeding a calcium-poor food, or an acid food, or a combination of the two, has resulted in a disease of the bone which was, in gross appearance, very similar to rachitis. Miwa and Stoeltzner[2] found that there were certain differences between this artificial disease of the bone and true rachitis. There was slight disturbance in the endochondral ossification and a very considerable "osteoporose" and Stoeltzner therefore called this artificial disease pseudo-rachitic osteoporosis.

Götting[3] and Schmorl confirmed these results. Clinically the rosary and enlarged epiphyses come in those cases in which there is a deficit of calcium in the bone. Such bone is more easily broken. It is a generally accepted fact that pseudorachitic osteoporosis comes as a result of a calcium-poor food; that it is the physiologic reaction of an organism with healthy bones; and that the bone substance is qualitatively normal but

1. Schmorl: Die pathologische Anatomie der rachitischen Knochenkrankung mit besonderer Berücksichtigung ihrer Histologie und Pathogenese. Ergebn. d. inn. Med. u. Kinderh., 1909, iv, 403.

2. Miwa and Stoeltzner: Ueber die bei jungen Hunden durch kalkarme Fütterung entstehende Knochenerkrankung, Beitr. z. path. Anat. u. allg. Path., 1898, xxiv, 578.

3. Götting: Ueber die bei jungen Tieren durch kalkarme Ernährung und Oxalsäurefütterung entstehenden Knochenveränderungen. Virchow's Arch. f. path. Anat., 1909, cxcvii, 1.

has quantitatively less calcium than the corresponding normal bone. This phase of the subject will be treated in more detail later.

An acid food causes the same microscopic picture as a food containing a diminished amount of calcium and must come into the same class as pseudorachitic osteoporosis.

Recently an attempt has been made to prove that some of the diseases of metabolism are due to a disturbance of the function of certain glands having an internal secretion. These are, respectively, the thyroid, parathyroid, thymus and adrenal glands. These attempts have not been successful.

Finally, rachitis has been considered an infectious disease. Schmorl has been unable to isolate any organism to which he could attribute rachitis, except in the case of two rats twenty-four hours old with a disease of the bone which was morphologically similar to a moderately severe rachitis. These observations corresponded with those of Morpurgo,[4] who found a diplococcus in the bones of young rats which were clinically rachitic. J. Koch[5] believes that negative findings are due to poor staining methods and the difficulty of demonstrating bacteria in bones. He was able to demonstrate bacteria both in culture and section in the epiphyses of ribs of children with rachitis who had died in the course of measles, diphtheria, pertussis, scarlet fever, gastro-enteritis, etc. The kind of bacteria found in his cases were often different, and most prominent among them were streptococci, bacteria coli, and staphylococci. In the majority of these cases the heart blood was sterile and the bacteria which were found in the epiphyses were not part of a general infection. He concludes with Kassowitz that rachitis is principally a vascular change of the epiphyses and adds that this change is due to bacterial infection of the part.

PATHOLOGY

The Calcification of Bone in Rachitis

Lehnerdt[6] summarizes the literature on this phase of the subject from seventy odd papers, the references for which will not be given in this review but may be found in the original.

According to Pommer, all newly formed bone first deposits osteoid material which later becomes calcified. This calcification goes on so

4. Morpurgo: Ueber eine infectiöse Form der Knochenbrüchigkeit bei weissen Ratten. Verhandl. d. Deutsch. path. Gesellsch., 1900, iii, 40; Beobachtungen an Serienschnitten von osteomalacischen und rachitischen Knochen. Verhandl. d. Deutsch. path. Gesellsch., 1909, xiii, 51.

5. Koch, J.: Untersuchungen über die Lokalisation der Bakterien, das Verhalten des Knochenmarkes und die Veränderungen der Knochen, insbesondere der Epiphysen, bei Infectionskrankheiten mit Bemerkungen zur Theorie der Rachitis. Ztschr. f. Hyg. u. Infectionskr., 1911, lxix, 436.

6. Lehnerdt: Warum bleibt das rachitische Knochengewebe unverkalkt? Ergeb. d. inn. Med. u. Kinderh., 1910, vi, 120.

quickly in health that the osteoid layer remains thin. In rachitis the characteristic picture is one with a broad osteoid layer which is the result of some interference with the deposit of lime salts.

Other writers believe that the abnormal amount of osteoid tissue in rachitis is due to the fact that calcium has been robbed from the bone which was once calcified, and others that all bone substance formed during the active or florid stage of rachitis was uncalcified.

A calcium deficit in the bones in rachitis may be attributed theoretically to the following causes:

I. A primary deficit of calcium in the food.

II. A secondary deficit of calcium, there being sufficient calcium in the food.

(*a*) It is either not absorbed in sufficient quantities for the needs of the growing bone, or

(*b*) It is absorbed in sufficient quantity but is excreted again in abnormal amounts; and

(1) Either considerable calcium is lost as a result of an abnormal digestion, or

(2) A sufficient quantity is absorbed into the organism, but for some reason cannot be used in the bone and therefore is excreted again.

I. PRIMARY CALCIUM DEFICIT

It has long been known that rachitic bones are poor in calcium and on the basis of this knowledge an attempt was made to produce artificial rachitis in animals by feeding them food low in calcium. These experiments caused clinical changes similar to those seen in rachitis and the question came up as to whether true rachitis was produced or not. Chemical analyses of these bones showed that they had a low ash and calcium content. Stoeltzner and Miwa's experiments showed that these changes were not outside of physiologic limits, and these writers concluded that they had to deal with a pseudorachitis giving it the name pseudorachitic osteoporosis. These bones, as mentioned above, show a low ash and calcium content, but they do not lose relatively as much magnesium as do the bones of true rachitis. In the former the loss of phosphoric acid goes hand in hand with the loss of calcium, while in the latter there is relatively less phosphorus lost than calcium.

The rosary and enlargement of the epiphyses comes in all cases in which there is a diminution in the deposit of calcium. Such bones are easily broken. Finally, in pseudorachitic osteoporosis the abnormally broad zone of growth cannot be demonstrated under the microscope and is always present in true rachitis. The two diseases are, therefore, best differentiated by the pathologist. On the basis of these facts Lehnerdt agrees with Stoeltzner and Miwa that pseudorachitic osteoporosis is a

result of a food containing a low amount of calcium, is the physiologic reaction of an organism with healthy bones to a calcium-poor food and that the bone substance is qualitatively normal and quantitatively diminished in amount. A deficit of calcium in the food, therefore, cannot cause true rachitis.

Aron[7] concludes that the normal infant takes only as much calcium from human milk as it requires for growth. Some of the cases in which rachitis develops on human milk are due to too small a percentage of calcium in the milk.

Bahrdt and Edelstein[8] found the average CaO in human milk to be 0.0426 per cent., while Schabad[9] found that the calcium content of human milk is greater at the beginning of nursing than at the end, and that there is a progressive diminution in the percentage of calcium during the course of lactation, falling off from 0.045 per cent. to 0.031 per cent. The decrease is constant and regular in some women and in others irregular and with great variations. The average percentage of calcium in human milk on which healthy babies were fed is 0.044 per cent. and rachitic babies 0.039 per cent. Rachitis is possible with a large percentage of calcium in the milk, even with 0.055 to 0.088 per cent. The milks on which children develop rachitis contain larger amounts of the organic constituents, for example, fat, than do the milks of healthy babies. The caloric value of the rachitogenic milk is therefore higher. For every 100 milk calories in rachitis there were 63 mg. of calcium and in health 76.5 mg. It is obvious, therefore, that the rachitic baby must either take the required amount of food calories containing too little calcium or to obtain the necessary quota of calcium it must take too much food. It is, according to Schabad, impossible to increase the amount of calcium in human milk. He believes that the clinical manifestations due to a calcium deficit in the food are not true rachitis but are pseudo-rachitic osteoporosis.

II. SECONDARY CALCIUM DEFICIT

Sufficient proof has not been obtained from complete metabolism experiments to warrant the conclusion that a decrease in the retention of calcium is responsible for its decreased absorption. Either the gastro-intestinal canal must be in such a condition that there is an increased excretion of calcium or the newly formed bone is in such condition that, during the active stages of rachitis, the calcium which has already been

7. Aron: Kalkbedarf und Kalkaufnahme beim Säugling und die Bedeutung des Kalkes für die Aetiologie der Rachitis. Biochem. Ztschr., 1908, xii, 28.

8. Bahrdt and Edelstein: Das Kalkangebot in der Frauenmilch. Jahrb. f. Kinderh., 1910, lxxii, Suppl., p. 16.

9. Schabad: Der Kalkgehalt der Frauenmilch. Zur Frage der ungenügenden Kalkzufuhr als Ursache der Rachitis. Jahrb. f. Kinderh., 1911, lxxiv, 511.

absorbed into the body cannot be deposited in the normal manner and is therefore reexcreted into the intestine.

Birk[10] and Rothberg found that there was a negative calcium balance (i. e., more calcium was excreted from the body than was ingested in the food) in those cases in which there were large amounts of fat in the food. In their whole milk experiments the ash balance was always negative, this being more marked in young children, and both these writers concluded that milk-fat had an unfavorable influence on the retention of ash, more especially calcium and magnesium. They believed, furthermore, that there was first an excess of fatty acids in the intestine and that these fatty acids withdrew the salts (among them calcium) from the body to form soaps. The calcium retention increased as the soap formation decreased. Birk concluded that rachitis was due to a primary disturbance of digestion. Lehnerdt considers that this conclusion is not based on sufficient proof, especially because the fatty acids can combine with only relatively small amounts of the alkaline salts. Bahrdt[11] considers that there were more bases excreted by the rachitic children reported by Birk, than could be combined with the soaps present. Freund[12] found that with an increase of fat and the formation of soap stools there was no diminution in the calcium balance.

Birk[13] performed a series of experiments on two healthy and two rachitic children to see if cod-liver oil had an unfavorable action on the digestion of fat and caused demineralization of the body. The action of the cod-liver oil and phosphorus caused a doubtful negative mineral balance to improve in the two rachitic children and had no effect on the healthy children because they retained only as much mineral salts as they needed in growth. The absolute amount of soaps in the stools of the rachitic children was considerably larger than that in the stools of the healthy children, while the percentage of soap in the stools of the rachitic children was reduced. Freund[14] commented on the fact that the absolute amount of soap in the stools of the healthy children was the same before and after the administration of phosphorus and cod-liver

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10. Birk: Ueber den Magnesiumsatz des Säuglings. Jahrb. f. Kinderh., 1907, lxvi, 300; Rothberg: Ueber den Einfluss der organischen Nährungskomponenten auf den Kalkumsatz künstlich genährter Säuglinge. Jahrb. f. Kinderh., 1907, lxvi, 69.

11. Bahrdt: Untersuchungen über das Symptom der Seifenbildung und die Ausscheidung der Basen im Darm des Säuglings. Jahrb. f. Kinderh., 1910, lxxi, 249.

12. Freund: Zur Wirkung der Fettdarreichung auf dem Säuglingstoffwechsel. Jahrb. f. Kinderh., 1905, lxi, 36.

13. Birk: Untersuchungen über den Einfluss des Phosphorlebertrans auf den Mineralstoffwechsel gesunder und rachitischer Säuglinge. Monatschr. f. Kinderh., 1908, vii, 450.

14. Freund: Physiologie und Pathologie des Fettstoffwechsels im Kindesalter. Ergebn. d. inn. Med. u. Kinderh., 1909, iii, 139.

oil and that in the rachitic children there was a lower percentage of soap and a larger amount of neutral fat and fatty acids when using phosphorus and cod-liver oil. If the soaps were responsible for the loss of calcium in rachitis the condition would be similar to that produced in animals and there would result not rachitis but pseudorachitic osteoporosis.

Lehnerdt apparently considers the following hypothesis of Stoeltzner most plausible — that the newly formed bone remains uncalcified because it is not in condition to impregnate itself with the calcium which is brought to it in the body fluids. When the calcium is rejected by the bone it must be excreted and returned to the intestine, where it combines with the fatty acids to form soaps. If this is true, calcium is the cause of the excessive formation of soaps instead of being a result of an abnormal fat digestion.

The acid theory of the pathogenesis of rachitis is without foundation because animals fed on acids do not develop rachitis and there is no diminution in the alkalinity of the blood.

Stoeltzner's[15] theory that the bone in rachitis is unable to deposit calcium in the normal manner is developed as follows: There are three stages in the development of bone: (1) the osteoid, in which the bone does not possess the power of depositing salts; (2) the potential, in which it obtains the power, and (3) the actual, in which calcium is deposited. If there is too little calcium in the food the stages progress in a normal manner except that the advance from the potential to the actual stage of deposited calcium should not take place because of the lack of calcium, and one should find abnormal amounts of osteoid tissue. This, however, is not the case. The old bone, which has already calcified, gives up its calcium to the new bone and the new bone continues to develop in the normal manner, passing from the potential to the actual stage. Thus the newly formed bone is constructed at the expense of the old bone because there is not enough calcium at hand to supply all the demands of the growing skeleton. The abnormally broad zone of osteoid tissue of true rachitis is not seen in experimental rachitis (pseudorachitic osteoporosis) because there is merely a deficit of calcium in the food, and not a condition which makes it impossible to deposit calcium.

Lehnerdt[16] experimented with animals and found by substituting strontium for calcium in the food, that bone changes only came when there was a deficit of calcium. The severity of strontium pseudorachitis

15. Stöltzner: Die zweifache Bedeutung des Calciums für das Knochenwachstum. Arch. f. d. ges. Physiol. (Pflüger), 1908, cxxii, 599.

16. Lehnerdt: Zur Frage der Substitution des Calciums im Knochensystem durch Strontium. I. Der Einfluss des Strontiums auf die intrauterine Entwicklung des Knochensystems. Beitr. z. path. Anat. u. z. allg. Path., 1909, xlvi, 468; II. Strontiumfütterung an säugende Tiere. Beitr. z. path. Anat. u. z. allg. Path., 1909, xlvii, 215; Phosphorsklerose und Strontiumsklerose, Jahrb. f. Kinderh., 1910, lxxii, 395, 610.

depends on the amount of the deficit. This condition is rapidly cured when calcium is again given in the food. Lehnerdt concludes, as a result of his own observations, that true rachitis is in all probability a disturbance of the intermediary metabolism and he considers that "domestication" plays an important rôle in the subject. Rachitis is not a disease of the free, wild animal, but is often found in animals confined in zoological gardens. Similarly the children of a pastoral race are apt to develop it when confined to a city.

v. Hausemann[17] considered that it resulted from too little air and activity. For example, those children born in the fall who are necessarily housed all winter in bad quarters and with poor ventilation had rachitis more frequently than children born in the spring and living under the same circumstances. Schmorl[1] found that in a great majority the disease commenced between the months of November and March. They tend to cure spontaneously during the summer months and to relapse in the fall. Ebbell's[18] attention was attracted to the question of why rachitis is a relatively rare disease in the tropics. A study of its geographical distribution shows that it increases in frequency the greater the distance from the equator, irrespective of race. He concludes that rachitis is the result of lack of light. This assumption was apparently confirmed by some experiments on animals and plants which were deprived of light and in which deformities developed. He thinks that the ultraviolet ray is particularly important and points out that this ray is partially arrested by window glass. Therefore children kept in the house are deprived of the ultraviolet ray in particular. Sittler[19] believes that it is a true disturbance of digestion which is caused in part by unhygienic surroundings, more especially during the winter months. Czerny[20] defines rachitis as a congenital constitutional anomaly whose most prominent symptom is the change in the bones and cartilages. There is evidently a general disturbance of nutrition affecting the bones and inducing symptoms on the part of the nervous system. Hutinel[21] regards a quantitative modification of the bone-marrow as the essential feature of rachitis. He thinks that the qualitative changes are similar to those encountered in ordinary infections; but the remarkable proliferation of the bone-marrow seems to be specific for rachitis. Marfan[22] says that one must admit that all

17. v. Hausemann: Ueber Rachitis als Volkskrankheit. Berl. klin. Wchnschr., 1906, xliii, 249.

18. Ebbell: Norsk Mag. f. Laegevidensk., 1909, lxix; abstr. in Jour. A. M. A., 1908, l, 1390.

19. Sittler: Neuere Ansichten über die Aetiologie der Rachitis. Fortschr. d. Med., 1909, xxvii, 817.

20. Czerny: Die erste Vorlesung in Strassburg, 9 Mai, 1910; Monatschr. f. Kinderh., 1910, ix, 131.

21. Hutinel: LeRachitisme. Arch. de méd. des enf., 1910, xiii, 81.

22. Marfan: Le Rachitisme et sa pathogenie bailliere et fils, Paris, 1911.

rachitis carry a focus of infection or chronic intoxication. Rachitis does not act like a special single disease but a syndrome in which bony deformity is the most prominent symptom.

The work of Schabad and his pupils on rachitis has been prominent in medical literature and has added much to our knowledge of normal and diseased bone. He[23] found that the dried bones of the newly born infant contained 60 to 65 per cent. of ash and 40 to 45 per cent. of organic material. The amount of ash in the bone varies from birth onward; at first the ash increases and at the second year decreases to 59 to 55 per cent. and during this time there is a corresponding increase in the organic material. The ash begins to increase at the end of the second year up to the adult 68 per cent. of the dried bone substance. The percentage of water in rachitic bone is higher and the percentage of dry substance is lower than in healthy bone. The relation of ash to organic matter is 20:80 in rachitic bone and 60:40 in healthy bone. Krasnogorski[24] also found a high water content in rachitic bones with a mineral content of one-half the normal. The calcium and magnesium were especially reduced. These facts left no doubt in the author's mind that the large amount of water in rachitic bones stands in causal relation to the poverty of calcium ions. The disturbance of the intracellular calcium metabolism must change the water content of the organism and have a powerful influence over the entrance of water into the organism. Under this influence the hyaline substance of the bone absorbs water and there results the clinical enlargement of the epiphyses of rachitis. Gassmann[25] concludes as a result of his analyses of healthy and rachitic bones that the complex salt $(Ca_2(PO_4)_2)3CaCO_3$ is the same in both instances. Potassium, sodium and chlorin are the same in both but magnesium is increased from 0.50 per cent. in normal bone to 0.53 to 0.74 per cent. in rachitic bones. He concludes, therefore, that magnesium plays an important part in the pathology of the disease.

Schabad[26] reports further that the weight of the skeleton during the first year of life is 16 per cent. of the body weight. The calcium content of the skeleton is 1.25 per cent. of the body weight and 7.7 per cent. of the skeletal weight. The greatest deposit of calcium takes place during the period of greatest growth, i. e., in a breast-fed baby between the second

23. Schabad: Zur Bedeutung des Kalkes in der Pathologie der Rachitis. Der Mineralgehalt gesunder und rachitischer Knochen. Arch. f. Kinderh., 1909, lii, 47.

24. Krasnogorski: Ueber die Wirkung der Ca-Ionen auf das Wasserabsorptionsvermögen der Knorpelgewebes und ihre Bedeutung in der Pathogenese des rachitischen Prozesses. Jahrb. f. Kinderh., 1909, lxx, 643.

25. Gassmann: Chemische Untersuchungen von gesunden und rachitischen Knochen. Hoppe-Seyler's Ztschr. f. physiol. Chem., 1910, lxx, 161.

26. Schabad: Zur Bedeutung der Kalkes in der Pathologie der Rachitis. II. Der physiologische Kalkbedarf und Rachitis infolge von unbefriedigtem Kalkbedarf. Arch. f. Kinderh., 1910, lii, 68.

and fourth months, and in a bottle-fed baby between the second and sixth months. The calcium retention of a baby taking human milk may be as high as 70 per cent. of the intake and one taking cows' milk 30 per cent. Although the calcium content of cows' milk is four times greater than that of human milk, the calcium needs during the period of greatest growth can only be furnished by two-thirds milk or richer, because the assimilation of cows' milk is two and one-half times poorer than human milk. A baby may take human milk containing deficient calcium for three months' time without any symptoms of rachitis, and therefore the connection between a low percentage of calcium in the food and rachitis is not established. Rachitis, on the other hand, may develop in a baby taking human milk containing the normal or a diminished amount of calcium. He agrees with Stoeltzner that experimental rachitis caused by feeding animals a calcium-poor food is clinically, anatomically and chemically indistinguishable from spontaneous rachitis, but that histologically it is characterized as a pseudorachitic osteoporosis. Furthermore, it is probable that in man there is a condition other than rachitis in which a deficient amount of calcium in the food causes a pseudorachitis which cannot be differentiated clinically from rachitis, but which has the characteristics of pseudorachitis.

In acute rachitis[27] and during the early or florid stage of the disease the calcium balance is either diminished or negative, and as the disease progresses and becomes well developed the calcium balance is either below normal or within normal limits. When convalescence commences there is increased retention of calcium which shows itself earlier than does clinical improvement. During this latter period two to three times more calcium is retained than is normal and when the cure is complete the calcium retention again becomes normal.

The increased excretion of calcium in progressive or florid rachitis goes on exclusively through the intestines and at the same time there is less calcium than usual excreted through the urine. Dibbelt[28] obtained the same results and believes that this is evidence against the theories of increased resorption of calcium from the bones or of inability of the rachitic osteoid tissue to take up calcium, because, according to him, they are not compatible with the decreased absorption of calcium in the urine. In either of these condition the urinary calcium should be increased. Schabad interprets these findings as evidence against the acid theory of rachitis.

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27. Schabad: Zur Bedeutung des Kalkes in der Pathologie der Rachitis: III. Der Kalkstoffwechsel bei Experimenteller. Arch. f. Kinderh., 1910, liii, 380.

28. Dibbelt: Weitere Beiträge zur Pathogenese der Rachitis. Verhandl. der deutsch. path. Gesellsch., 1910, xiv, 294.

The relation between calcium and phosphorus was then studied by Schabad[29] and he found that there was an increased excretion of phosphorus as well as calcium in the acute stage of rachitis. There was relatively so much phosphorus excreted that it could not all have come from the bone and presumably was lost from the phosphorus-rich nervous system. The relation between the phosphorus in the urine and feces in nurslings is 80 : 20 in health and 65 : 35 in rachitis; and in the bottle-fed baby 60 : 40 in health and 40-44 : 60-56 in rachitis. During convalescence from rachitis the total excretion of phosphorus is lower than normal and the relation between the phosphorus in the urine and the feces returns to the normal figures. The following connection has been established between the calcium and phosphorus excretion: An increased excretion of calcium causes a greater retention of phosphorus, and vice versa. The great loss of phosphorus is important in the disease and may explain the nervous symptoms. Birk and Orgler[30] found that a disturbance of the calcium metabolism may be present quite a while before the clinical appearance of rachitis. Orgler[31] criticizes Schabad's deductions and does not consider them free from error. In one instance he was able to increase the calcium retention by changing the quality of the food, and in another instance an increase in the amount of calcium in the food did not influence the calcium retention at all. Orgler believes that there are two important conditions which regulate the assimilation of calcium: (1) the power of the body to assimilate it, which depends on the intermediary metabolism concerning which we know nothing; and (2) the composition of the food. Aschenheim and Kaumheimer[32] examined the muscles of eight children with rachitis and three without rachitis and found that in all cases of rachitis there was a diminution in the amount of calcium which apparently corresponded with the severity of the disease. Bing[33] found excessive thinning of the muscle fibers with loss of striation accompanied by an increase of the muscle-cell nuclei without interstitial infiltration. He found these changes less marked in milder forms of rachitis and never found the same condition in the control muscles examined.

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29. Schabad: Zur Bedeutung des Kalkes in der Pathologie der Rachitis: IV. Der Phosphorstoffwechsel bei Rachitis. Arch. f. Kinderh., 1910, liv, 83.

30. Birk and Orgler: Der Kalkstoffwechsel bei Rachitis. Monatschr. f. Kinderh., 1910, ix, 544.

31. Orgler: Ueber den Kalkstoffwechsel bei Rachitis. Monatschr. f. Kinderh., 1911, x, 373.

32. Aschenheim and Kaumheimer: Ueber den Aschegehalt der Muskulatur bei Rachitischen. Monatschr. f. Kinderh., 1911, x, 435.

33. Bing: Histopathologische und Elektrodiagnostische Untersuchungen bei rachitischen Kindern mit pseudoparetischen und atonischen Muskelstörungen. Med. Gesellsch., Basel, Dec. 6, 1906: Reported in Cor.-Bl. f. Schweiz. Aerzte, 1907, xxxvii, 119.

Findlay[34] examined the blood in rachitis and summarized his findings as follows: In active and uncomplicated rickets anemia is not the rule but is to be regarded as exceptional, and when it occurs is due to adventitious causes. On the contrary, the amount of hemoglobin and the number of red cells in rachitic children in the series examined are notably in excess of the normal average. The red blood corpuscles, as a rule, vary more in size than in normal individuals of similar ages, but otherwise there is no abnormality. The nucleated red cells, polychromatophils and myelocytes are of the rarest occurrence. In rickety subjects there is little characteristic change as far as the leukocytes are concerned. They may be normal, slightly increased, or even diminished in number. The mononuclears more frequently than the polynuclears show an absolute increase in the number per cubic millimeter.

TREATMENT

Kassowitz[35] first recommended phosphorus in the treatment of rachitis. His original prescription, known as phosphorlebertan (phosphori 0.01, ol. jecor aselli, ad 250), still holds first place in Germany in the treatment of rachitis. Kissel[36] found in his experiments on animals that phosphorus had absolutely no effect on the skeletal system, and concluded that there was no ground for its use. Despite this report phosphorus continues to be used and many experiments have been performed to prove its efficiency.

Birk[37] and Schabad[38] both concluded that phosphorus in therapeutic doses does not affect the calcium metabolism in healthy children. Such children take only as much phosphorus as they need for growth regardless of the amount in the food. In rachitis cod-liver oil increases the retention of phosphorus and calcium and this action is intensified by the addition of phosphorus to the oil.[39] The increased retention of calcium starts three to five days after giving phosphorus and gradually diminishes until at the end of two months it is again normal. This depends on the increased absorption and decreased excretion through the urine and feces. The question now comes up as to whether oils as such in combination with

34. Findlay: The Blood in Rickets. Lancet, London, 1909, i, 1164.

35. Kassowitz: Die Phosphorbehandlung der Rachitis. Ztschr. f. klin. Med., 1883-4, vii, 36.

36. Kissel: Ueber die pathologisch-anatomischen Veränderungen in den Knochen wachsender Tiere unter dem Einfluss minimaler Phosphordosen. Virchow's Arch. f. path. Anat., 1896, cxliv, 94.

37. Birk: Untersuchungen über den Einfluss des Phosphorlebertrans, etc. Monatschr. f. Kinderh., 1908-09, vii, 450.

38. Schabad: Der Phosphor in der Therapie der Rachitis. Der Einfluss des Phosphors auf den Kalkstoffwechsel bei rachitischen und gesunden Kindern. Ztschr. f. klin. Med., 1909, lxvii, 454.

39. Schabad: Die Behandlung der Rachitis mit Lebertran, Phosphor und Kalk. Ztschr. f. klin. Med., 1909, lxviii, 94.

phosphorus have a therapeutic action on rachitis. Schabad[40] investigated the action of phosphorus, cod-liver oil and "sesamol" on the metabolism of calcium, phosphorus, fat and nitrogen and found that "sesamol" and phosphorus did not help rachitis, while cod-liver oil plus phosphorus increased the retention of phosphorus and calcium and the absorption of fat and nitrogen. Schabad and Sorochowitsch[41] used lipanin, i. e., olive a substitute for cod-liver oil, which was supposed to be easily absorbed because it contained free fatty acids. They concluded from their metabolism experiments that lipanin and olive oil increase the absorption of nitrogen and fat but that lipanin has no advantage over olive oil. Lipanin does not inerease the retention of calcium in rachitis and is therefore not as good as cod-liver oil in the treatment of rachitis. They say in their most recent article[43] that sometimes phosphorus and cod-liver oil does not have a favorable action on the retention of calcium in rachitis, especially if the disease is not approaching a convalescence. At other times they have a favorable action on the calcium retention. They experimented with various other salts combined with cod-liver oil and found that a calcium acetate cod-liver oil had the most favorable action on rachitis because it contained much more calcium.

Most recently Caroline Towles[44] did a series of metabolism experiments in von Pirquet's clinic in Breslau and was unable to demonstrate that phosphorus cod-liver oil had any action at all on acute rachitis. The most recent work of Towles and Schabad and Sorochowitsch indicates, therefore, that phosphorus cod-liver oil may not have any therapeutic action in the treatment of acute rachitis.

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40. Schabad: Phosphor. Lebertran und Sesamöl in der Therapie der Rachitis. Ihr Einfluss auf den Kalk-, Phosphor-, Stickstoff-, und Fettstoffwechsel. Ztschr. f. klin. Med., 1909-10, lxix, 435.

41. Schabad and Sorochowitsch: Lipanin als Ersatzmittel des Lebertrans bei Rhachitis. Sein Einfluss auf den Stoffwechsel. Monatschr. f. Kinderh., 1911, ix, 659.

oil with the addition of 6 per cent. oleic acid according to v. Mering,[42] as

42. v. Mering: Ein Ersatzmittel für Lebertran. Therap. Monatsch., 1888, ii, 49.

43. Schabad and Sorochowitsch: Die Behandlung der Rachitis mit Lebertranemulsionen und ihre Einwirkung auf den Stoffwechsel. Monatschr. f. Kinderh., 1911, x, 12.

44. Towles, C.: Untersuchungen über den Einfluss des Phosphorlebertrans auf den Stoffwechsel eines rachitischen Säuglings. Ztschr. f. Kinderh., 1910-11, i, 346.

INDEX OF CURRENT PEDIATRIC LITERATURE

ANATOMY, PHYSIOLOGY AND HYGIENE

Baby-Tents of Chicago. F. W. Allin.
Journal A. M. A., Dec. 30, 1911.

Biologic Differentiation of Albuminoids in Milk. (Die biologische Differenziering der Milcheiweisskörper.) H. Kleinschmidt.
Monatschr. f. Kinderh., 1911, x, No. 8.

Butter in Human Milk. (Etude du beurre dans de lait de femme par la centrifugation.) E. Plauchu and R. Rendu.
Lyon Médical, Nov. 19, 1911.

Clean Milk from Producer's Standpoint. H. E. Van Norman.
Pennsylvania Med. Jour., December, 1911.

Determination of Actual Size of Infants. (Ueber eine Methode zur Bestimmung des Volumes bei Säuglingen.) K. Oppenheimer.
Ztschr. f. Kinderh., 1911, iii, No. 3.

Development of Acidity in Cows' Milk and Its Relation to Time and Temperature. R. Vincent.
Glasgow Med. Jour., December, 1911.

Epidemic of Sore Throat Occurring in Boston and Vicinity During May, 1911. J. L. Goodale.
Boston Med. and Surg. Jour., Dec. 14, 1911.

Epidemic of Tonsillitis Due to Infected Milk. M. W. Richardson.
Boston Med. and Surg. Jour., Dec. 14, 1911.

Improved Medical Inspection of Public Schools and Its Results. G. J. Holmes.
Jour. Med. Soc. New Jersey, December, 1911.

Instruction of Teachers in School Hygiene. G. M. Whipple.
Woman's Med. Jour., December, 1911.

Medical Inspection in Public Schools, Its Needs and Desirabilities. A. B. Montgomery.
Jour. Oklahoma Med. Assn., December, 1911.

Medical Inspection in Toledo Public Schools. Its Aims and Some of Its Results. P. B. Brockaway.
Am. Med. Compend, December, 1911.

Medical Inspection of Schools. G. E. Hyde.
Northwest Medicine, December, 1911.

Medical Inspection of Schools. R. W. Jones.
Wisconsin Med. Jour., November, 1911.

The Menstrual Cycle, Its Physiology and the Preparation of the Young Girl for Its Advent. E. S. Brown.
Woman's Med. Jour., November, 1911.

Milk and Its Relation to Public Health. P. J. Eaton.
Pennsylvania Med. Jour., December, 1911.

Need of Medical Inspection of School Children in Ireland. O. S. Gogarty.
Dublin Jour. Med. Sc., December, 1911.

Outbreak of Tonsillitis or Septic Sore Throat in Eastern Massachusetts and its Relation to Infected Milk Supply. C. E. A. Winslow.
Boston Med. and Surg. Jour., Dec. 14, 1911.

Physiologic Differences Between the Cortex and the Medulla of the Thymus. (Bedeutung der eosinophil-gekörnten Blutzellen im menschlichen Thymus.) H. Schridde.
München. med. Wchnschr., Dec. 5, 1911.

Physiology of Lactation. (Bedarf des physiologischen Reizes zur Anregung und Erhaltung der Laktation?) H. Helbich.
Monatschr. f. Kinderh., 1911, x, No. 8.

Preservation of Health in School Children. R. G. Freeman.
Med. Rec., New York, Dec. 16, 1911.

Sanitary Control of New York's Milk Supply. E. J. Lederle.
Pennsylvania Med. Jour., December, 1911. Pediat., December, 1911.

Tentative Classification of Exceptional Children. M. P. E. Croszmann.
Cal. Jour. Med., December, 1911.

PATHOLOGY AND BACTERIOLOGY

Amyotrophy plus Obesity in Young Children; Two Cases. (Zur Frage der früinfantilen Amyotrophen und einer sie begleitenden "endogen-" Fettsucht.) T. Gott and H. Schmidt.
Ztschr. f. Kinderh., 1911, iii, No. 3.

Atrophy of Certain Muscles as Cause of Lordotic Albuminuria. (Juvenile Dystrophie und lordotische Albuminurie im Kindesalter.) E. Ebstein.
Ztschr. f. Kinderh., 1911, iii, No. 3.

Cancer of Ovary in Girl of Five. (Krebs des Eierstocks bei einem fünfjährigen Mädchen; Ovariotomie.) W. W. Rosanoff.
Deutsch. med. Wchnschr., Dec. 14, 1911.

Carcinoma and Sarcoma in Early Childhood. Q. W. Hunter.
Pediat., December, 1911.

Causes of Limping in Children. D. Savariaud.
Med. Press and Circular, Dec. 13, 1911.

Congenital Deformities of the Arms and Hands. (Ueber angeborene Verbildungen im Bereiche der oberen Extremität.) P. Glaessner.
Deutsch. med. Wchnschr., Dec. 14, 1911.

Cultural Studies on Mouth Spirochætæ. (*Treponema Microdentium* and *Macrodentium*.) H. Noguchi.
Jour. Exper. Med., January, 1912.

A Dicephalic Monster. F. W. Bullen.
Jour. Minn. Med. Assn., Dec. 15, 1911.

Dull and Backward Children. A. F. Tredgold.
Med. Press and Circ., Nov. 29, 1911.

Large Umbilical Hernia, Containing Omentum, Gut and Liver, in Child Seven Months Old; Operation, Recovery. E. S. Allen.
Kentucky Med. Jour., Nov. 15, 1911.

No Basis for Assumption that Cretinism is Transmissible. (Zur Kontaktinfektion des Kretinismus.) A. Flinker.
Wien. klin. Wchnschr., Dec. 21, 1911.

Precociously Developed Male Pseudohermaphrodite. (Frühreifer Scheinzwitter.) R. Asch.
Berl. klin. Wchnschr., Dec. 25, 1911.

Reaction Curve in Glycerin Broth as Aid in Differentiating Bovine from Human Type of Tubercle Bacillus. M. Grund.
Jour. Med. Research, December, 1911.

Recent Studies in Experimental Pathology of Acute Anterior Poliomyelitis. P. A. Lewis.
Pennsylvania Med. Jour., December, 1911.

Recurrent Jaundice, Pyrexia, Splenomegaly, Anemia and Pigmentation of Skin in Girl Aged 11 Years. L. Guthrie.
Practitioner, December, 1911.

Relative Importance of Bovine and Human Types of Tubercle Bacilli in Different Forms of Human Tuberculosis. W. H. Park and C. Krumwiede.
Jour. Med. Research, December, 1911.

Research on the Stomach in Tetany in Children. (Magenuntersuchungen bei Kindlicher Tetanie.) F. Leist.
Monatschr. f. Kinderh., 1911, x, No. 8.

Significance of Enlarged Lymph-Nodes in Children. G. K. Varden.
Atlanta Jour.-Rec. Med., December, 1911.

Simultaneous Contrast Stain for Diphtheria Bacilli. (Eine neue einzeitige Doppelfärbungsmethode für die Polkörperchen der Diphtheriebazillen.) M. Raskin.
Deutsch. med. Wchnschr., Dec. 21, 1911.

Spasms Nutans; Report of Four Cases. H. K. Hill.
Arch. Pediat., December, 1911.

METABOLISM AND NUTRITION

The Ash Content of the Muscles with Rachitis. (Ueber den Aschgehalt der Muskulatur bei Rachitischen.) E. Aschenheim and L. Kaumheimer.
Monatschr. f. Kinderh., 1911, x, No. 8.

Breast Milk for Sick Infants. (Kranke Kinder bei Ernährung mit Frauenmilch.) L. Langstein and E. Hoerder.
Therap. Monatschr., December, 1911.

Calcium Metabolism in Infantile Tetany, with Report of a Case. H. Schwarz and M. H. Bass.
Am. Jour. Dis. Child., January, 1912.

Capillary Test of Viscosity of Milk. (Vergleichende Viskositätsbestimmungen der Milch mit Hilfe ihrer kapillären Steighöhe.) A. Kreidl and E. Lenk.
Wien. klin. Wchnschr., Nov. 30, 1911.

Diabetes Mellitus in Childhood. W. Gellhorn.
Northwest Med., December, 1911.

Fatal Case of Infantile Scurvy Following Diet Confined Almost Exclusively to "Grape Nuts." J. W. Amesse.
Denver Med. Times and Utah Med. Jour., December, 1911.

Feeding of Children. D. E. English.
Jour. Med. Soc. New Jersey, December, 1911.

Infant Feeding. C. H. Johnston.
Physician and Surg., November, 1911.

Infant Feeding *Au Naturel*. S. Delano.
Boston Med. and Surg. Jour., Jan. 11, 1912.

Nutrition and Metabolism. H. A. Milliken.
Jour. Maine Med. Assn., January, 1912.

Nutrition of Children. S. F. Hale.
Southern Med. Jour., December, 1911.

Practical Infant Feeding. A Study of 400 Cases. R. A. Benson.
Am. Jour. Obst. and Dis. of Women and Child., December, 1911.

Quantitative Analysis of Human and of Cow's Milk. A. V. Meigs and H. L. Marsh.
Med. Rec. New York, Dec. 30, 1911.

Recent Experiences in the Artificial Feeding of One Hundred Infants During the First Three Months of Life. F. C. Neff.
Jour. Am. Med. Assn., Dec. 23, 1911.

Salvarsan in Scorbutus. (Ueber Salvarsanbehandlung bei Skorbut.) M. Tuschinsky and G. Iwaschenzow.
München. med. Wchnschr., Dec. 12, 1911.

Splenomegaly (Gaucher). M. S. Reuben.
Am. Jour. Dis. Child., January, 1912.

The "Split Proteid" Method of Artificial Feeding. W. D. Boggs.
Cal. Jour. Med., December, 1911.

Studies on Infant Nutrition: II. The Hard or Casein Curds in Infants' Stools. A. M. Courtney.
Am. Jour. Dis. Child., January, 1912.

DISEASES OF THE NEWLY BORN

Congenital Fistulas in the Neck; Twenty-Three Cases. (Om medfødte halsfistler og endel med disse beslegtede anomalier.) F. Leegaard.
Norsk. Mag. f. Lægevidensk., December, 1911.

Congenital Imperforate Anus, with Occlusion of Entire Colon. C. H. Hamilton.
Jour. Am. Med. Assn., Dec. 30, 1911.

Fatal Jaundice of New-Born. Five Cases in One Family. J. N. Morris.
Australian Med. Jour., Oct. 21, 1911.

Glycocoll in the Urine of New-Born Infants. (Vorkommen von Glykokoll im Harn des Neugeborenen.) A. v. Reuss.
Ztschr. f. Kinderh., 1911, iii, No. 3.

The Prematurely Born. (Quelques considérations sur les diverses variétés de prématurés.) A. Hergott.
Ann. de gynéc. et d'obst., December, 1911.

Resuscitation of Asphyxiated Infants by the Insufflation Method of Meltzer and Auer. E. Plauchu.
Am. Jour. Dis. Child., January, 1912.

Sepsis from Bednar's Aphthæ in New-Born Infants. (Sepsis bei Neugeborenen ausgehend von den Bednar'schen Aphthen.) G. Linzenmeier.
Zentralbl. f. Gynäk., Dec. 16, 1911.

Spina Bifida. B. B. Cates.
Jour. Tennessee Med. Assn., December, 1911.

Two Cases of Intracranial Cerebral Hemorrhage in New-Born Relieved by Operation. C. C. Simmons.
Boston Med. and Surg. Jour., Jan. 11, 1912.

The Ultimate Fate of the Prematurely Born. (Avenir éloigné du prématuré.) V. Wallich and A. Fruhinsholz.
Ann. de gynéc. et d'obst., November, 1911.

ACUTE AND INFECTIOUS DISEASES

Acute Anterior Poliomyelitis. B. F. Green.
Jour. Michigan Med. Soc., December, 1911.

Acute Poliomyelitis: Diagnosis. D. Hecht.
Interstate Med. Jour., December, 1911.

Autoserotherapy in Infectious Diseases. P. Modinos.
Presse méd., Dec. 6, 1911.

Cancrum Oris. Case Following Typhoid Fever. J. R. Lockhart.
Mississippi Med. Month., December, 1911.

Case of Mild Anterior Poliomyelitis. W. N. Bullard.
Boston Med. and Surg. Jour., Jan. 11, 1912.

Cerebrospinal Meningitis as Recurrence After Salvarsan. (Zerebrospinalmeningitis als Rezidiv nach Salvarsan.) E. Oberholzer.
München. med. Wchnschr., Dec. 12, 1911.

Control of Measles. P. B. Brooks.
Med. Rec., New York, Dec. 23, 1911.

Diagnosis and Medical Treatment of Poliomyelitis. W. G. Spiller.
Pennsylvania Med. Jour., December, 1911.

Diphtheria Antitoxin and Anaphylaxis. J. A. Roddy.
New York Med. Jour., Dec. 16, 1911.

Diphtheria Carriers and Medical Inspection of Schools. H. Albert.
Iowa Med. Jour., December, 1911.

Epidemic Anterior Poliomyelitis in South Derbyshire. J. H. Moir.
British Med. Jour., Dec. 30, 1911.

Epidemic Paralysis in Hampshire. G. F. England.
British Med. Jour., Dec. 30, 1911.

Epidemic Poliomyelitis. A. D. Young.
Jour. Oklahoma Med. Assn., December, 1911.

Epidemic Poliomyelitis. L. W. Rork.
Western Med. Rev., December, 1911.

Epidemic Poliomyelitis Occurring at Stowmarket, Suffolk. S. Hillier.
Brit. Med. Jour., Dec. 30, 1911.

Experimental Research on Acute Poliomyelitis. II. (Maladie de Heine-Medin.) K. Landsteiner, C. Levaditi and M. Pastia.
Ann. de l'Inst. Pasteur, November, 1911.

The Kidney in Scarlet Fever. (Zur Frage der Scharlachnephritis.) (C. Leede.
München. med. Wchnschr., Nov. 28, 1911.

Pennsylvania's Work on Poliomyelitis. S. G. Dixon.
Pennsylvania Med. Jour., December, 1911.

Poliomyelitis. T. M. Koon.
Jour. Michigan Med. Soc., December, 1911.

Practical Points in Diagnosis and Treatment of Diphtheria. A. Levinson.
Med. Record, Jan. 6, 1912.

Prophylaxis and Treatment of Infantile Paralysis. J. W. Amesse.
Pediat., December, 1911.

Prophylaxis of Infectious Diseases in Infant Wards. (Die Verhütung und Bekämpfung der Infektionskrankheiten in geschlossenen Säuglingsanstalten.) J. Cassel.
Deutsch. med. Wchnschr., Nov. 30, 1911.

Rheumatism in Children. T. M. Anderson.
Am. Jour. Obst., December, 1911.

Smallpox and Chickenpox. H. W. Hill.
Journal-Lancet, Jan. 1, 1912.

Treatment of Diphtheria Carriers by Overriding Infected Area with Culture of Staphylococcus Pyogenes Aureus. H. Page.
New York Med. Jour., Dec. 23, 1911.

Two Cases of Poliomyelitis Mistaken for Neuritis. T. A. Williams.
Boston Med. and Surg. Jour., Jan. 11, 1912.

Unusual Case of Typhoid in Young Girl. F. J. Barrett.
Med. Rec., New York, Dec. 16, 1911.

TUBERCULOSIS AND SYPHILIS

Household Syphilis (Syphilis Economica), with Observations on Acquired Syphilis in Infants. J. A. Nixon.
Bristol Med.-Chir. Jour., December, 1911.

Present Status of Our Knowledge of Infectiousness of Milk Containing Tubercle Bacilli. P. C. Heinemann.
Illinois Med. Jour., December, 1911.

Prognosis of Tuberculosis in Young Infants. (Zur Prognose der tuberkulösen Infektion im frühen Säuglingsalter.) J. Ibrahim.
Beitr. z. Klin. d. Tuberk., 1911, xxi, No. 2.

Syphilis as Factor in Abnormal Development of Children. E. W. Dittrich.
Am. Jour. Obst., January, 1912.

Tubercular Laryngitis in Infant of Seventeen Months. J. M. Hunt.
Med. Press and Circular, Dec. 6, 1911.

Tubercular Peritonitis in Children with Special Reference to Fibrous Form. J. M. Acker.
Mississippi Med. Month., December, 1911.

Tuberculin Reactions and Their Comparative Value as Diagnostic Aids. O. H. Benker.
Interstate Med. Jour., December, 1911.

DIGESTIVE SYSTEM

Albumin Milk as Contrasted with Other Milk Mixtures in Treatment of Diarrheas of Children. H. Heiman.
Arch. Pediat., December, 1911.

Care of Children's Teeth. J. L. Austin.
Jour. Tennessee Med. Assn., December, 1911.

Infantile Hypertrophic Stenosis of Pylorus, Based on Seven Operated Cases. F. E. Bunts.
Am. Jour. Med. Sc., January, 1912.

Molds in Alimentary Canal. I. P. Hall.
Canadian Med. Assn. Jour., December, 1911.

Pyloric Stenosis in Infants. J. B. Bilderbach.
Med. Sentinel, December, 1911.

Summer Diarrhea in Infants and Children. M. J. Synnott.
Arch. Pediat., December, 1911.

Type of Nervous Vomiting in Childhood. E. B. Smith.
Lancet, London, Dec. 23, 1911.

Volvulus. E. P. Magruder.
Surg., Gynec. and Obst., December, 1911.

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Case of Catarrhal Laryngitis Requiring Intubation. J. T. Ullom.
Arch. Pediat., December, 1911.

Drainage Tube in Pleural Cavity. (Removed with Aid of Fluoroscopic Screen.) F. Huber.
Arch. Pediat., December, 1911.

The Treatment of Pertussis with Vaccine. E. E. Graham.
Am. Jour. Dis. Child., January, 1912.

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Heart in Acute Affections. C. G. Jennings.
Jour. Michigan Med. Soc., December, 1911.

Leukemia in Childhood. W. F. Cheney.
Am. Jour. Med. Sc., January, 1912.

NERVOUS SYSTEM

Causes of Convulsion in Infancy and Childhood. E. Pritchard.
Clinical Journal, Nov. 22, 1911.

Chorea of Genital Origin. (Chorea genitalen Ursprunges.) L. M. Bossi.
Zentralbl. f. Chir., Dec. 9, 1911.

Management of the Nervous Child. M. L. Perry.
Jour. Kansas Med. Soc., November, 1911.

Meningeal and Cerebral Symptoms in Infants. (Meningeale und cerebrale Krankheitsbilder beim Neugeborenen und Säugling.) W. Knöpfelmacher.
Med. Klinik, Dec. 24, 1911.

Nervous Diseases in Children. J. P. Parkinson.
Clinical Journal, Nov. 29, 1911.

Nervous Manifestations in Children and Their Treatment. R. O. Moon.
Med. Press and Circ., Dec. 20, 1911.

Overfeeding and Convulsions. (Ueberfütterung und Krämpfe.) E. Ebstein.
Ztschr. f. Kinderh., 1911, iii, No. 3.

The Recognition of the Pre-Neurasthenic and Pre-Insane Condition in the Young. A Study for the General Practitioner. R. Moore.
Jour. Am. Med. Assn., Dec. 23, 1911.

Report of Brain Cases. W. J. Butler.
Am. Jour. Dis. Child., January, 1912.

GENITO-URINARY SYSTEM

Enuresis and Thyroid Extract. A. C. D. Firth.
Lancet, Dec. 9, 1911.

Primary Pyelitis in Infants. J. T. Fotheringham.
Canadian Med. Assn. Jour., December, 1911.

Treatment of Gonorrheal Cervicitis. (Zur Behandlung der Cervicalgonorrhöe.) Wagner.
Berl. klin. Wchnschr., Dec. 25, 1911.

Venereal Diseases and Their Relation to Infant Mortality and Race Deterioration. P. A. Morrow.
New York Med. Jour., Dec. 30, 1911.

EYE, EAR, NOSE AND THROAT

Are the Tonsils a Menace or a Protection? H. L. Swain.
Ann. of Oto., Rhin., and Laryng., September, 1911.

Cases of Late Secondary Hemorrhage after Tonsillotomy. F. E. Hopkins.
Ann. of Oto., Rhin. and Laryng., September, 1911.

Cases of Night-Blindness with Peculiar Conjunctival Changes in Children. S. Stephenson.
Brit. Jour. Child. Dis., December, 1911.

Civic Medical Inspection of School Children, With Special Reference to Diseases of Eye, Ear and Throat. S. C. Ayres.
Lancet-Clinic, Dec. 23, 1911.

Clinical Aspects of Epidemic of Septic Sore Throat in Cambridge, May, 1911. E. A. Darling.
Boston Med. and Surg. Jour., Dec. 14, 1911.

Complications Following Tonsillectomy. I. A. Lederman.
Kentucky Med. Jour., Dec. 15, 1911.

Condition of Nose, Throat and Ear as Factor in Exceptional Development. O. Glogau.
Am. Jour. Obst., January, 1912.

Indications for and Method of Removal of Tonsils. J. A. Duff.
West Virginia Med. Jour., December, 1911.

Multiple Papillomata of Larynx in Children. E. C. Sewell.
Cal. Jour. Med., January, 1912.

Phase of Tonsil Question. R. W. Perry.
Northwest Med., December, 1911.

Postoperative Tonsillar Hemorrhage. J. E. Sawtell.
Jour. Kansas Med. Soc., November, 1911.

Relation of Enlarged Tonsils to Endocarditis. A. C. Getchell.
Ann. of Oto., Rhin., and Laryng., September, 1911.

Some Popular Misconceptions Regarding Ophthalmia Neonatorum. E. M. Alger.
New York State Jour. Med., December, 1911.

Strabismus. (Ueber Entstehung und Behandlung des Schielens.) C. Adam.
Med. Klin., Dec. 17, 1911.

Technic of Tonsillectomy. J. M. Ingersoll.
Ann. of Oto., Rhin., and Laryng., September, 1911.

Tonsils in Pathology. (Die Tonsillen als Eintrittspforte der Infektionskrankheiten.) G. Finder.
Med. Klin., Dec. 10, 1911.

Tonsil Instruments. R. J. Tivnen.
Illinois Med. Jour., December, 1911.

Why Re-Operate in Adenoids? G. W. Spohn.
Jour. Indiana Med. Assn., December, 1911.

SURGERY

Certain Operative Procedures in Paralysis of Children. R. Jones.
British Med. Jour., Dec. 9, 1911.

Pin in Bronchus—Report of a Case. F. G. Hodgson.
Atlanta Jour.-Rec. Med., December, 1911.

A Point in the Treatment of Lateral Curvature of the Spine. E. A. Rich.
Jour. Am. Med. Assn., Dec. 30, 1911.

Simple Appliance for the Relief of Residual Peroneal Paralyses Following Acute Anterior Poliomyelitis. C. O. Griffin.
Therap. Gaz., December, 1911.

Surgery and Electro-Therapeusis in Infantile Paralysis: Attempt to Define Sphere of Each.
British. Jour. Child. Dis., December, 1911.

Surgical Treatment of Poliomyelitis. G. G. Davis.
Pennsylvania Med. Jour., December, 1911.

MISCELLANEOUS

Backward Child vs. the Feeble-Minded Child. M. P. E. Groszmann.
Am. Jour. Obst., January, 1912.

Care of the Crippled and Rachitic in Italy. D. C. McMurtrie.
Med. Rec., New York, Dec. 16, 1911.

Clinical Studies of Exceptional Children. E. B. McCready.
Am. Jour. Obst., January, 1912.

Common Ailments in Infancy and Their Treatment. E. Pritchard.
Pediat., December, 1911.

Etiologic Factors in Exceptional Children and Their Prevention. M. Neustaedter.
Am. Jour. Obst., January, 1912.

Progressive Methods of Dealing with Juvenile Delinquency. N. B. Neelen.
Am. Jour. Obst., January, 1912.

Providing for Crippled Children. C. J. Cook.
Lancet-Clinic, Dec. 9, 1911.

Social and Moral Considerations Related to Medical and Surgical Care of Crippled Children. D. C. McMurtrie.
New York Med. Jour., Dec. 30, 1911.

Speech Defects in Children. J. M. Fletcher.
Am. Jour. Obst., January, 1912.

Unnecessary Infant Mortality. G. H. Irvin.
Cleveland Med. and Surg. Reporter, November, 1911.

Value of Physical Activities in Treatment of Atypical Boys. G. Meylan.
Am. Jour. Obst., January, 1912.